Cybersickness: A Visuo-Vestibular Multisensory Integration Approach

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Declaration of Authorship

I, Maria Gallagher, hereby declare that this thesis and the work presented in it is entirely my own. Where I have consulted the work of others, this is always clearly stated.

Signed: _____

Date: _____

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My educational journey has been quite a strange one over the years! From home education, to college where I was adamant I was ***never*** going to university… rapidly followed by a BSc., MSc., and finally the PhD thesis you see here. It's funny how things work out! I'm so very lucky to have been supported by a plethora of amazing people along the way, and I doubt I could fully do justice to all of them in my acknowledgements section, but to everyone who has backed me on my educational journey so far, thank you!

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"Miss Tick sniffed. 'You could say this advice is priceless,' she said. 'Are you listening?'
'Yes,' said Tiffany.
'Good. Now… If you trust in yourself…'
'Yes?'
'…and believe in your dreams…'
'Yes?'
'…and follow your star…' Miss Tick went on.
'Yes?'
'…you'll still get beaten by people who spent their *time working hard and learning things and weren't so lazy. Goodbye.'"*

- Terry Pratchett, *The Wee Free Men*

Abstract

Virtual Reality (VR) has grown in popularity in recent years. Applications range from recreation and gaming, to training, education, and rehabilitation. Although technological improvements are stark since the first VR headsets, a troubling problem remains where up to 80% of VR users will experience debilitating symptoms of nausea, disorientation, headaches and fatigue. This cybersickness therefore remains a significant barrier to VR uptake. Sensory conflict appears to be the likely cause of cybersickness. In typical VR scenarios, vision signals that the user is moving through the virtual environment while the vestibular system signals that the user is stationary. Thus, in order to adapt to the VR environment and reduce cybersickness the brain must re-weight vestibular cues for self-motion. While this multisensory re-weighting may reduce cybersickness, vestibular processing may be significantly altered following adaptation to the virtual environment. Such VR after-effects have not been extensively explored.

The main thesis of my PhD is that in order to adapt to VR sensory conflict, the central nervous system re-weights vestibular sensory information in accordance with principles of sensory cue integration. This vestibular re-weighting reduces cybersickness during VR exposure, but may entail alterations in vestibular sensory processing after exposure. First, I outline a framework of VR adaptation based on optimal multisensory integration models, in which vestibular cues are re-weighted during and after exposure to visual cues for self-motion in VR. According to this framework, I then explore how vestibular processing at both the perceptual and physiological level is altered by exposure to self-motion in VR. Next, I investigate methods of cybersickness prevention based on vestibular down-weighting and sensory augmentation through artificial vestibular stimulation (Galvanic Vestibular Stimulation, GVS). Finally, I also quantify the natural equivalent of GVS-induced self-motion in order to finesse the technique for cybersickness prevention. Overall, these findings highlight the key role of visuo-vestibular multisensory integration in VR, describing previously unknown after-effects of VR exposure and providing potential future avenues for cybersickness reduction.

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Chapter 1:

Introduction

The popularity of Virtual Reality (VR) has grown rapidly in recent years. By 2022, it is believed that commercially available VR headsets will attract over 315 million users worldwide and will represent an economy worth over $80 billion (Bellini et al., 2016). Thus, VR represents a substantial market with considerable economic impact. The uses of VR are extensive and varied, from widely-known entertainment and gaming applications, to education and training (Pelargos et al., 2017), rehabilitation (da Silva Cameirão, Bermúdez Badia, Duarte, & Verschure, 2011; Rose, Nam, & Chen, 2018; Viñas-Diz & Sobrido-Prieto, 2016) and research (Macauda et al., 2015; Russo et al., 2017). In recent decades, technological improvements in VR headsets have been vast, with improved resolution, display frame rates, and field of view, resulting in increased realism of the virtual environment (Ranadive, Harsora, Khanvilkar, & Sayyad, 2017; Shafer, Carbonara, & Korpi, 2019). However, despite these improvements in technological factors, a troublesome problem remains where between 20 and 80% of VR users will experience worrying symptoms of nausea, disorientation, and oculomotor disturbances termed *cybersickness* (Cobb, Nichols, Ramsey, & Wilson, 1999; Munafo, Diedrick, & Stoffregen, 2017; Rebenitsch & Owen, 2016; Stanney, Kennedy, & Drexler, 1997).

Cybersickness can be regarded as a form of motion sickness induced by exposure to immersive VR (Kennedy, Drexler, & Kennedy, 2010; Mazloumi Gavgani, Walker, Hodgson, & Nalivaiko, 2018; Rebenitsch & Owen, 2016). Typically, symptoms can include nausea, fatigue, eyestrain, headaches, and blurred vision, although it has been reported that the most severe symptoms are related to disorientation (Kennedy et al., 2010; Kennedy, Lane, Berbaum, & Lilienthal, 1993;

Rebenitsch & Owen, 2016). In addition, physiological changes such as increases in heart rate, skin conductance, and tachygastric power have been associated with cybersickness (Dennison, Wisti, & D'Zmura, 2016; Mazloumi Gavgani, Nesbitt, Blackmore, & Nalivaiko, 2017; Kim, Kim, Ko, & Kim, 2001). These symptoms tend to increase over time during VR exposure (Liu, 2014; Moss et al., 2011; Stanney, Kingdon, Graeber, & Kennedy, 2002).

Individual factors which can influence cybersickness development include gender (Chen, Chao, Chen, Wang, & Tan, 2015; Flanagan, May, & Dobie, 2005; Stanney, Hale, Nahmens, & Kennedy, 2003), age (Arns & Cerney, 2005), balance control (Weech, Varghese, & Barnett-Cowan, 2018) and previous history of motion sickness (Nichols, 2000; Rebenitsch & Owen, 2014). However, at present it is not possible to fully and reliably predict which VR users are more likely to experience cybersickness. Prevention of cybersickness has been a key aim for VR developers, but currently there is limited evidence regarding the most effective methods. Repeated exposure (Hill & Howarth, 2000; Howarth & Hodder, 2008), software and hardware modifications (such as overlaid visual references for gravity, Chang et al., 2013; Han et al., 2011, or improved positional tracking, Llorach, Evans, & Blat, 2014), and artificial vestibular stimulation (Cevette et al., 2012; Reed-Jones, Reed-Jones, Trick, & Vallis, 2007; Weech, Moon, & Troje, 2018) have all shown promise in reducing cybersickness. However, these potential solutions have not had widespread adoption, as they may require access to specialist equipment or entail additional side effects for certain users (Utz et al., 2011). Accordingly, further research into both predictive factors for cybersickness and methods of cybersickness prevention is necessary.

As well as cybersickness, exposure to VR can also lead to after-effects following return to the real world. It has been reported that symptoms of disorientation

can be up to 95 times higher one hour following VR exposure (Stanney & Kennedy, 1998). In addition, proprioceptive coordination is poorer, with individuals having difficulties coordinating the eyes, head, and hands after exposure to VR (Harm, Taylor, Reschke, Somers, & Bloomberg, 2008; Stanney, Kennedy, Drexler, & Harm, 1999). Moreover, changes in vestibular reflexes are also apparent following VR. Specifically, vestibulo-ocular reflex gain decreases substantially after exposure to VR, approaching recovery only after 30 minutes (Di Girolamo et al., 2001). The full extent of VR after-effects is not known, and a complete account of their time-course and underlying causes is yet to be established.

The precise causes of cybersickness are debated (Rebenitsch & Owen, 2016; Riccio & Stoffregen, 1991), however it seems likely that it may arise from sensory conflicts induced by exposure to VR (Bos, Bles, & Groen, 2008; Reason & Brand, 1975; Rebenitsch & Owen, 2016). Under normal circumstances, vision and the vestibular system provide coherent cues regarding the direction and speed of self-motion. However, during exposure to VR, vision signals that the user is moving in a certain direction at a certain speed, while the vestibular system signals that the user is stationary. This conflict between visual and vestibular cues for self-motion may therefore be the underlying cause of cybersickness (Bonato, Bubka, & Palmisano, 2009; Bos et al., 2008; Keshavarz & Hecht, 2011; Reason & Brand, 1975).

Self-motion in the real world depends on the integration of visual, proprioceptive, and vestibular cues (Greenlee et al., 2016). These latter cues are particularly important for self-motion perception (Britten, 2008; DeAngelis & Angelaki, 2012; Dichgans & Brandt, 1978). The vestibular organs are located inside the inner ear and comprise of three orthogonal semicircular canals (anterior, posterior, and lateral) and two otolith organs (saccule and utricle). The semicircular canals detect

angular rotations of the head in roll, pitch and yaw, while the otolith organs detect linear acceleration from both translation and gravity. Accordingly, the integration of these vestibular signals provides a comprehensive representation of the head in 3D space (Cullen, 2019; Glover, 2004). Crucially, patients who have experienced bilateral vestibular loss do not experience symptoms of motion sickness, suggesting a key role for the vestibular system in motion sickness in general, and more specifically cybersickness (Cheung, Howard, & Money, 1991; Paillard et al., 2013).

In the past two decades evidence has suggested that visuo-vestibular integration for self-motion follows the predictions of Bayesian frameworks for multisensory integration (Ernst & Banks, 2002; Ernst & Bülthoff, 2004). Specifically, perception of heading direction is more precise when both visual and vestibular cues are available, compared to the unimodal estimates (Angelaki, Gu, & DeAngelis, 2011; DeAngelis & Angelaki, 2012; Gu, Angelaki, & DeAngelis, 2008). In addition, as the reliability of one cue decreases, the weighting placed on the other cue increases (Angelaki et al., 2011; DeAngelis & Angelaki, 2012; Gu et al., 2008). Moreover, this re-weighting process can occur dynamically in an adaptive response to fluctuating cue reliability (Fetsch, Turner, DeAangelis, & Angelaki, 2009).

We therefore proposed that adaptation to VR may follow a dynamic multisensory re-weighting process, accounting for reductions in cybersickness and after-effects of VR (Gallagher & Ferrè, 2018). During VR exposure, visual cues signal that the user is moving in a certain direction with a certain acceleration, namely vection. However, corroborating vestibular cues are absent, instead signalling that the user is stationary. Exposure to visuo-vestibular conflicts such as this likely underlies development of cybersickness symptoms (Bonato et al., 2009; Bos et al., 2008; Keshavarz & Hecht, 2011; Rebenitsch & Owen, 2016). To adapt to the conflict and

enjoy the VR experience, the unreliable vestibular cues for self-motion may be down-weighted, while visual cues are up-weighted. Accordingly, self-motion perception comes predominantly from vision, while inputs from the vestibular system are minimised. Thus, the saliency of the visuo-vestibular conflict is reduced, lessening cybersickness symptoms. On return to self-motion in the real world, vestibular cues are once again present, for instance when the user walks around. The brain must therefore go through a further process of re-weighting, such that vestibular cues are up-weighted. It is in this period of re-weighting that after-effects of VR exposure may occur. Previously reported changes in disorientation and vestibulo-ocular reflex gain may provide evidence for this framework (Di Girolamo et al., 2001; Stanney & Kennedy, 1998).

In my PhD thesis I will detail and investigate the visuo-vestibular multisensory integration perspective on cybersickness. The overarching hypothesis is that the central nervous system dynamically re-weights vestibular sensory cues for self-motion during and after exposure to visuo-vestibular conflict in VR. Specifically, when visual cues signal that the user is moving while the vestibular system signals that the user is stationary, for example in a VR driving simulator, vestibular cues for self-motion are down-weighted such that cues for self-motion are predominantly extracted from vision. This vestibular down-weighting reduces the visuo-vestibular conflict, resulting in lower cybersickness. On return to the real world, where both vestibular and visual cues for self-motion are both available, an up-weighting of vestibular cues occurs, potentially accounting for VR-induced after-effects. Importantly, the work described in this thesis systematically explores different predictions of the multisensory integration framework, utilising a range of multidisciplinary methods and techniques, including cognitive neuroscience, vestibular physiology, and virtual reality research.

My work investigated whether exposure to vection in VR impacts vestibular processing at both perceptual and physiological levels, based on changes in dynamic vestibular re-weighting. Crucially, I show for the first time that conscious detection of vestibular signals is significantly worse following exposure vection in VR. Importantly, this may not be a mere bias or non-specific effect: I demonstrated that exposure to vection in VR also dramatically modulates low-level vestibular reflexes, i.e. a gold-standard physiological proxy for the processing of afferent vestibular signals. My results highlight the significant impacts of VR on vestibular processing. Importantly, while previous research has shown changes in vestibular functioning following 20 minutes of exposure to VR (Di Girolamo et al., 2001), my work demonstrates how changes to both perceptual and physiological vestibular processing can occur surprisingly rapidly, after less than five minutes of VR exposure.

In addition to the effect of VR on vestibular processing, my research has also focused on developing theory-driven strategies to prevent cybersickness based on the visuo-vestibular multisensory integration framework. Firstly, I have investigated whether down-weighting vestibular cues by reducing their reliability could reduce cybersickness. While previous research has investigated this through artificially stimulating the vestibular system (Weech, Moon, et al., 2018), I investigated whether this could be achieved simply by modifying body orientation with respect to gravity. In this posture, the otolith organs can no longer reliably signal the position of the head with respect to the gravitational vector (Vimal, DiZio, & Lackner, 2017), resulting in reduced vestibular weighting and increased visual weighting (Alberts et al., 2016; Ward, Bockisch, Caramia, Bertolini, & Tarnutzer, 2017). Secondly, I also investigated whether matching visual and vestibular cues for self-motion could prevent visuo-vestibular conflict, resulting in reduced cybersickness. To do this, I developed an

integrated Galvanic Vestibular Stimulation (GVS)+VR application. GVS involves artificially stimulating the vestibular nerve through small direct currents applied to the mastoids. This results in a sensation of roll rotation towards the cathode (Day & Fitzpatrick, 2005; Fitzpatrick & Day, 2004). Accordingly, GVS was applied during left and right turns during the GVS+VR application, matching visual and vestibular cues for self-motion. While these two mechanisms are likely to require more fine-tuning to significantly reduce symptoms, they highlight potential future avenues for cybersickness prevention techniques.

Finally, having realised the limitations of the current knowledge on artificial vestibular stimulation technology, I have addressed a non-trivial theoretical and technical question: what is the perceived natural equivalent of GVS-induced self-motion? This has not only the potential to increase our understanding of vestibular functioning but also to precisely re-couple visual and artificial vestibular cues in future VR applications. Here I showed for the first time a quantifiable perceptual equivalent between natural and artificial vestibular cues. Thus, these findings are likely to be of significant value for those investigating GVS as a method of cybersickness prevention.

These studies and theoretical review confirm that visuo-vestibular conflicts in VR have clear consequences for the VR user experience. Symptoms of cybersickness and VR-induced after-effects remain a significant and poorly understood problem, despite clear technological advancements in VR technology in recent decades. To prevent adverse outcomes of VR exposure and significantly improve VR enjoyment, visuo-vestibular conflicts must be minimised. By following the visuo-vestibular multisensory integration perspective, negative impacts of VR exposure can be clearly identified, and theoretically driven solutions proposed. In resolving these problems, the full potential of VR applications can be realised.

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Chapter 2:

Methods

My work focuses on a visuo-vestibular multisensory integration perspective for cybersickness (Chapter 3), and investigates predictions derived from this framework. Specifically, I explored whether vestibular processing was altered by exposure to vection in Virtual Reality (VR) (Chapters 4 and 5), and whether techniques based on disrupting visuo-vestibular conflicts could reduce cybersickness (Chapters 6 and 7). I also investigated whether these techniques could be refined by improving our theoretical understanding of artificial vestibular stimulation (Chapter 8). To address these research questions, I used a range of multidisciplinary methods, including vestibular methods, VR, and psychophysics. Specific experimental protocols are described in depth in each respective chapter. However, this chapter aims to provide a broad overview of the general methods used.

Vestibular Methods

Galvanic Vestibular Stimulation

Galvanic Vestibular Stimulation (GVS) is a safe, well controlled, and widely used method of artificially stimulating the vestibular system. Electrodes placed on the mastoids send a small direct current to stimulate the vestibular nerve (Curthoys & MacDougall, 2012; Kim, 2013; Stephan et al., 2005). While debate is ongoing, it seems likely that GVS activates both otolith and semicircular canal afferents (Cohen, Yakushin, & Holstein, 2012; Curthoys & MacDougall, 2012; Kwan, Forbes, Mitchell, Blouin, & Cullen, 2019). GVS was used in Chapter 4 to investigate vestibular perceptual processing following VR, and in Chapter 7 as part of an integrated

GVS+VR driving scenario to reduce cybersickness. A quantification of the illusory motion sensation elicited by GVS was conducted in Chapter 8 by using a psychophysical approach to estimate the equivalence between natural motion and the illusory GVS sensation.

In all three of these chapters, I used a binaural-bipolar GVS configuration with a boxcar waveform. This configuration involves placing an anode on one mastoid and a cathode on the other, with anodal currents decreasing the firing rate of the nerve and cathodal currents increasing them (Goldberg, Smith, & Fernández, 1984). Recently, Kwan et al. (2019) described vestibular afferent responses to sinusoidal binaural-bipolar GVS in primates. Importantly, otolith and semicircular canal afferents responded comparably to one another, with increased gain and phase lead in function of the frequency of stimulation. Moreover, irregular afferents were more responsive to GVS than regular afferents, with higher gains across GVS frequencies. Interestingly, responses to GVS and natural motion differed in that both regular and irregular semicircular canal afferents and irregular otolith afferents displayed greater high-pass tuning and phase leads in response to natural rotation versus GVS. Finally, despite the differences in firing rate to GVS, both regular and irregular semicircular canal and otolith afferents showed similar neuronal detection thresholds to GVS (Kwan et al., 2019). GVS therefore results in a polarity-dependent virtual roll rotation vector in which the participant experiences a sensation of roll rotation towards the cathode (Cathers, Day, & Fitzpatrick, 2005; Fitzpatrick & Day, 2004; Fitzpatrick, Marsden, Lord, & Day, 2002).

While GVS can be used to explore wider effects of vestibular activation on a particular task (Ferrè, Berlot, & Haggard, 2015; Mast, 2009; Volkening et al., 2014), here the illusory rotation sensation itself was of interest. Specifically, asking

participants to detect whether they felt the GVS-induced roll rotation during exposure to vection in VR provided evidence that perceptual vestibular processing was impaired after exposure to plane-congruent visual stimuli in VR (Chapter 4). In addition, the sensation of illusory rotation was used to replace vestibular signals which would otherwise have been absent in an integrated GVS+VR driving scenario, providing a potential mechanism for improvement of cybersickness symptoms (Chapter 7). Finally, quantifying the illusory rotation evoked by GVS provided valuable information for further refinement of artificial vestibular stimulation techniques to reduce cybersickness in VR (Chapter 8). Accordingly, quantifying the illusory sensation allows VR developers to more precisely match self-motion sensations elicited by both vision and GVS, minimising conflict between the two modalities.

The configuration of GVS in my work limits the illusory sensation to roll rotation only. However more complex configurations have been recently described (Aoyama, Iizuka, Ando, & Maeda, 2015; Cevette et al., 2012). For instance, four-pole GVS, which involves placing electrodes on the temples in addition to the mastoids may result in illusory rotations on other axes, such as pitch or yaw (Aoyama et al., 2015). This particular configuration of GVS may therefore be advantageous when the illusory motion must closely match the visual self-motion signals presented in VR. For instance, a similar configuration was used to provide complex sensations of roll, pitch, and yaw in a flight simulator, reducing simulator sickness (Cevette et al., 2012). Thus, while the binaural-bipolar configuration was sufficient for the purposes of the experiments presented here, more complex forms of GVS could be useful for expanding the current findings.

In addition to the configuration of the GVS electrodes, the virtual rotation vector elicited by GVS is dependent on the intensity of the stimulation (Day &

Fitzpatrick, 2005). Specifically, higher intensities of GVS result in a greater sensation of rotation (Day & Fitzpatrick, 2005; Wardman, Day, & Fitzpatrick, 2003). Accordingly, in Chapter 4 we used 0.7 mA GVS stimulation to induce near-threshold sensations of roll rotation, while in Chapter 7, we used 1 mA GVS, which resulted in a clearer percept of self-motion during the GVS+VR driving simulator. In addition, the GVS virtual roll rotation was quantified at 1 mA and 2.5 mA in Chapter 8, confirming that greater sensations of rotation are felt at higher GVS intensities. Considerable variation in individual thresholds to GVS have been reported in the literature (Ertl, Klimek, Boegle, Stephan, & Dieterich, 2018; Kerkhoff et al., 2011; Oppenländer et al., 2015). For example, individual thresholds ranged from 0.4 to 1.5 mA in a study by Oppenländer et al. (2015), while Ertl et al. (2018) recently reported an average GVS threshold as high as 1.8 mA. Given this variability, it may be the case that participants in our studies varied somewhat in their perception of the stimulation, with some participants perceiving more rotation than others. This may be evidenced by the fact that there was a small amount of variability in the natural equivalent motion measured between participants in Chapter 8. To eliminate individual differences in GVS perception, several studies have used thresholding procedures to administer a sub-threshold stimulation (Kerkhoff et al., 2011; Oppenländer et al., 2015). Typically, the intensity of GVS is adjusted in steps of 0.1 mA until the participant reports no sensations of motion. While this technique is advantageous when the rotation sensation is undesirable in an experiment, here the illusory motion was the focus of the studies. Accordingly, thresholding may have been difficult to implement, given that participants may struggle to describe their experience of the illusory motion. Thus, we controlled for the GVS dose received by participants, such that they received the same intensity of stimulation within each study.

In addition to the illusory rotation sensation, GVS can also induce non-vestibular specific cutaneous sensations at the skin surface. To control for this, and other non-specific effects such as the knowledge that an unusual stimulation is occurring, we administered a sham stimulation. A variety of sham-controls have been reported previously (Utz, Keller, Kardinal, & Kerkhoff, 2011; Wilkinson, Nicholls, Pattenden, Kilduff, & Milberg, 2008). For instance, GVS electrodes can be placed on the mastoids but no current delivered (Wilkinson et al., 2008), or only a brief GVS pulse administered before turning off stimulation (Utz et al., 2011). However, these forms of sham may not necessarily control for the cutaneous sensations, while administering brief GVS pulses may still elicit a sensation of rotation, which would be unsuitable for the present experiments. Thus, the sham stimulation presented here involved placing electrodes on the base of the neck, approximately 5cm below the mastoid electrodes, to administer the same intensity of stimulation as the active GVS (Ferrè et al., 2015; Ferrè, Day, Bottini, & Haggard, 2013; Lopez, Lenggenhager, & Blanke, 2010). While this sham stimulation elicits similar cutaneous sensations, it does not elicit any sensations of rotation, effectively controlling for non-specific effects of GVS.

Natural Vestibular Stimulation

In Chapter 8, we investigated the natural equivalent motion to the illusory sensations elicited by GVS. Accordingly, we combined GVS with natural vestibular stimulation evoked by rotation on a 3D turntable to estimate at what velocity the real and artificial vestibular signals were cancelled. The 3D turntable consists of a chair mounted on three motorised axes, with the participants' head positioned at the intersection of these axes. Participants can be passively rotated in roll, pitch, and yaw,

either alone or in combination for more complex patterns of rotation. Importantly, the direction, acceleration, and velocity of rotation are completely defined by the researcher, such that the motion profile of the 3D turntable is precisely controlled (Ertl & Boegle, 2019).

In Chapter 8, we used a motion profile consisting of an initial velocity step of $20^\circ/s^2$ until a desired velocity (from 0.5-15°/s) was reached, followed by a constant acceleration ramp of 1°/s. This motion profile was chosen to avoid semicircular canal adaptation to constant acceleration (Goldberg & Fernandez, 1971; St George, Day, & Fitzpatrick, 2011). In addition, the motion profile mimicked previously described postural responses evoked by GVS (Wardman, Day, et al., 2003). Thus, the natural and artificial vestibular stimulation sensations felt similar.

Despite the clear advantage of controlling the motion profile of the 3D turntable, one problem with natural vestibular stimulation is that it may be difficult to control for non-vestibular sensations triggered by other senses. For instance, proprioceptive and somatosensory signals may be conveyed through the chair through vibrations or air moving across the body, or by pressure exerted on one side of the body as the turntable rotates (Ertl & Boegle, 2019). In order to minimise these extra-vestibular sensations, we ensured that padding was placed around the participants' legs, and relatively low velocities of motion were used. However, it is important to consider that these alternative sensations could not be completely eliminated.

Vestibular-Evoked Myogenic Potentials

As well as vestibular perceptual processing, I also investigated whether vestibular physiological processing would be altered by exposure to VR in Chapters 5

and 7. The vestibular system is implicated in a number of vestibulo-ocular and vestibulo-spinal reflexes (Angelaki & Cullen, 2008; Cathers et al., 2005; Cullen, 2010), many of which are examined in standard clinical assessments of vestibular functioning. Accordingly, to explore vestibular physiological processing, I measured cervical vestibular-evoked myogenic potentials (VEMPs) after exposure to VR.

VEMPs are vestibulo-collic reflexes, evoked by loud, high-frequency tone-burst sounds (Rosengren & Colebatch, 2018). Sound waves stimulate the saccule, activating the inferior vestibular nerve and transmitting to the lateral vestibular nucleus, medial vestibulospinal tract and the sternocleidomastoid muscle, culminating in a characteristic biphasic EMG response, i.e. a p13-n23 wave (Colebatch, Halmagyi, & Skuse, 1994; Rosengren & Kingma, 2013). VEMPs stimulation has also been shown to activate wider cortical vestibular regions, including the posterior insula, inferior parietal cortices, and middle and superior temporal gyri (Schlindwein et al., 2008). Interestingly, characteristics of VEMPs, such as amplitude or asymmetry, may be associated with differences in susceptibility to motion sickness (Fowler, Sweet, & Steffel, 2014; Tal et al., 2013).

To investigate vestibular functioning after exposure to vection in VR, VEMPs were elicited by placing electrodes on the sternocleidomastoid muscles and measuring the EMG response to air-conducted sounds. VEMPs were recorded using eVEMPs software and hardware (BioMed, Jena, Germany). Trials lasted 80ms, and were recorded at 2000 Hz sampling frequency. Details regarding amplification and filtering of the signal was not provided by the eVEMP software. Amplification of the EMG signal would ideally be conducted with a gain of 2000 (Rosengren et al., 2019), however values between 500 (Fowler et al., 2014) and 5000 (Tal et al., 2013) have previously been reported. Bandpass filtering between 5-1500 Hz is typically used,

ensuring that the main frequency component of VEMPs (40-60Hz) falls within this window (Rosengren et al., 2019). This filtering is necessary to remove artefacts, for example from electrical noise, movement, or perspiration (Huigen, Peper & Grimbergen, 2002), and improve signal to noise ratio. Trials were only included in the final VEMP average if muscle activity fell within 129 and 400 µV RMS and electrode impedance was less than 20 kΩ. Trials with artefacts were thus rejected from inclusion in the final average. The sound stimuli were 500 Hz tone-bursts presented at 100 dB sound-pressure level, with a duration of 7ms. One-hundred trials were averaged to give the final VEMP response, which was recorded automatically by software. Although VEMPs are normally used to assess vestibular peripheral functioning, one widely cited problem with their use in clinics and reported in research literature is the lack of standardisation of recording protocols (Rosengren, Colebatch, Young, Govender, & Welgampola, 2019). Importantly, to address this issue I used the same recording parameters with all participants in each experiment which used VEMPs, thus ensuring responses were comparable across conditions and studies. The parameters used also fall within recently recommended guidelines for VEMPs recording (Rosengren et al., 2019).

Overall, VEMPs are a gold-standard assessment of otolith functioning, while remaining non-invasive and easy to administer (Rosengren et al., 2019; Rosengren & Kingma, 2013; Venhovens, Meulstee, & Verhagen, 2016). Thus, they were a pragmatic way of assessing vestibular physiological processing during VR exposure. However, it is important to consider that VEMPs assess only otolith functioning, potentially limiting findings. Accordingly, future research could consider a battery of vestibular tests, such as measurement of vestibulo-ocular reflexes (Di Girolamo et al.,

2001) or postural responses (Wardman, Taylor, & Fitzpatrick, 2003), to explore other aspects of vestibular processing after VR exposure.

Virtual Reality

Head-Mounted Displays

In Chapters 4-7, VR stimuli were presented on an Oculus Rift CV1 or DK2 head-mounted display (HMD). While HMDs are the most readily-available commercial VR format, other types of VR are available, such as Computer-Aided Virtual Environments (CAVEs) which project the VR environment on the walls of a room, or virtual environments presented on computer screens or desktops (LaViola, 2000; Rebenitsch & Owen, 2016; Sharples, Cobb, Moody, & Wilson, 2008). Although all of these VR types may lead to visuo-vestibular conflicts, subtle differences in levels of cybersickness have been reported (Rebenitsch & Owen, 2014; Sharples et al., 2008). Specifically, HMDs may produce greater levels of sickness than other display types (Sharples et al., 2008). Despite these differences, it is likely that the visuo-vestibular multisensory integration framework remains applicable to other types of VR, beyond HMDs. However, a direct exploration of this was not possible in the thesis. Thus, these findings must be extended in future work to explore other VR formats.

Virtual Environments

To address the research questions in the thesis, it was necessary to use a combination of vection-inducing optic flow stimuli as well as full virtual environments. Optic flow stimuli were used to isolate the effects of vection on vestibular processing in Chapters 4 and 5. These stimuli consisted of a field of white

dots on a black background which either expanded, creating the sensation of forward vection, or rotated, creating the sensation of roll vection. Both stimuli included a fixation cross at the centre of the display, which has been shown to increase the sensation of vection in contrast to optic flow stimuli without fixation points (Becker, Raab, & Jürgens, 2002; Riecke, Schulte-Pelkum, Avraamides, & Bülthoff, 2004). In addition, we also ensured that the optic flow stimuli filled the entire field of view of the HMD, given that optic flow which covers a greater area of the visual field typically elicits stronger vection than smaller displays (Keshavarz, Riecke, Hettinger, & Campos, 2015; Palmisano, Mursic, & Kim, 2017). Importantly, we asked participants to verify that they felt the sensation of vection in both Chapters 4 and 5. However, we did not consider other factors, such as intensity, onset, or duration of vection. Considerable variability has been reported regarding the best methods for assessing the intensity of vection, including moving a joystick (Riecke et al., 2004), subjective ratings (Keshavarz, Hettinger, Kennedy, & Campos, 2014), and indicating when a particular distance had been travelled (Becker et al., 2002). While the relation between the experience of vection and subsequent vestibular processing may be an interesting avenue for future research, it was beyond the scope of the present thesis.

Here, we used optic flow stimuli which moved at a constant velocity on a single axis, in order to explore the effects of this simple vection on vestibular processing. These stimuli are similar to those used in previous studies which have reported an inhibition of vestibular cortical regions during optic flow (Brandt, Bartenstein, Janek, & Dieterich, 1998; Kleinschmidt, 2002) and to those which have investigated the effect of vestibular stimulation on optic flow sensitivity (Edwards, O'Mahonys, Ibbotson, & Kohlhagen, 2010; Holten & MacNeilage, 2018; Shirai & Ichihara, 2012). However, more complex optic flow stimuli, for example varying in velocity or including motion

on multiple axes, has been shown to increase activity in vestibular cortical regions (Kirollos, Allison, & Palmisano, 2017; Uesaki & Ashida, 2015). This may not be surprising, given that the vestibular system primarily detects fast changes in head motion, with vestibular responses declining during constant velocity motion (Fernández & Goldberg, 1976; Goldberg & Fernandez, 1971; St George et al., 2011; Waespe & Henn, 1977). Accordingly, it may be possible that the effects on vestibular processing following exposure to simple optic flow may differ when using more complex displays, potentially limiting the findings of the present thesis to this form of visual stimulus. However, it is important to consider that complex optic flow may entail greater conflict between visual and vestibular cues, which may result in more pronounced changes in vestibular functioning than those reported here. Given that many VR applications are likely to include complex visual motion, including rotations on multiple axes and changes in acceleration, the results of the present thesis must be extended to these types of stimuli.

To assess techniques to reduce cybersickness, more complex and longer duration VR environments were necessary. In Chapter 6, we used a custom VR rollercoaster to explore the effects of reduced vestibular reliability on cybersickness, while in Chapter 7 we used a custom integrated GVS+VR driving simulator to explore the effects of vestibular sensory substitution on cybersickness. Both scenarios were presented for approximately 10 minutes. As cybersickness symptoms typically accumulate over time (Kennedy, Drexler, & Kennedy, 2010; Liu, 2014), it is possible that longer exposure to these scenarios could have elicited greater levels of sickness. However, this presentation time was chosen predominantly to ensure participant comfort given the cybersickness interventions chosen.

Both VR scenarios used in Chapters 6 and 7 were passively observed by participants. Passive scenarios were selected in order to ensure all participants were exposed to the same visual stimuli across conditions. However, it is possible that active and passive VR scenarios may produce differences in levels of cybersickness (Sharples et al., 2008; Stanney & Hash, 1998). Specifically, lower cybersickness has been reported with active scenarios versus passive ones (Sharples et al., 2008; Stanney & Hash, 1998), potentially due greater predictability of sensory outcomes (Reason & Brand, 1975) or increased presence (Weech, Kenny, & Barnett-Cowan, 2019). Given that many commercial VR scenarios are likely to be actively controlled by VR users, the findings in the present thesis should also be explored in these active scenarios.

Cybersickness Measures

Typically, cybersickness is assessed through subjective questionnaires. Accordingly, we used the Simulator Sickness Questionnaire (SSQ; Kennedy, Lane, Berbaum, & Lilienthal, 1993) and Fast Motion Sickness Scale (FMS; Keshavarz & Hecht, 2011) to assess participants' symptoms in Chapters 6 and 7. Although originally derived to assess simulator sickness in military populations (Kennedy et al., 1993), the SSQ is the most widely used subjective questionnaire to assess cybersickness (Kennedy et al., 2010; Rebenitsch & Owen, 2016). It consists of 16 symptoms which participants are required to rate on a scale of 0 to 3, with higher scores corresponding to greater sickness. These symptoms are divided into three main clusters of nausea, disorientation, and oculomotor disturbances. Thus, the questionnaire gives scores for each of these three subscales, as well as an overall sickness score. Although the SSQ may be considered a standard measure of cybersickness, recent reports have suggested alternative factor groupings to account for cross-correlation between the three

subscales, differences between simulator sickness and cybersickness, and different respondent demographics (Bouchard, Robillard, & Renaud, 2007; Bruck & Watters, 2011; Kim, Park, Choi, & Choe, 2018). For instance, Kim et al. (2018) suggested that the SSQ could be reduced to 9 items, with only oculomotor and disorientation clusters in order to assess cybersickness following exposure to VR. By contrast, Bouchard et al. (2007) suggested that the SSQ should instead consist of oculomotor and nausea clusters, while Bruck and Watters (2011) proposed four new factors of general cybersickness, fatigue, arousal and vision. Thus, given these discrepancies and the fact that they are likely to require validation and wider employment, the established SSQ scoring was used in the present studies.

While the SSQ provides an overview of a range of symptoms experienced by participants, it cannot capture how symptoms develop across VR exposure. Accordingly, we used the FMS to explore the time-course of cybersickness in Chapter 6. The FMS requires participants to verbally rate their level of nausea on a scale from 0 to 20 every 60 seconds, where 0 is no sickness at all and 20 is frank sickness. Participants are instructed to focus on symptoms of nausea, stomach problems, and discomfort, but to ignore other symptoms such as nervousness, tiredness and boredom. Importantly, the FMS has good correspondence with the nausea subscale of the SSQ and has previously been used to assess cybersickness (D'Amour, Bos, & Keshavarz, 2017; Keshavarz & Hecht, 2011; Keshavarz, Hecht, & Zschutschke, 2011). However, given that it assesses only symptoms of nausea, we opted also to include the SSQ following VR exposure to fully capture participants' cybersickness in Chapter 6.

Like many questionnaire techniques, both the SSQ and FMS have the potential to be confounded by demand characteristics. For instance, participants may exaggerate or downplay symptoms, according to their view of the experiment. Importantly,

Young, Adelstein, and Ellis (2007) reported that demand characteristics for the SSQ were apparent when the questionnaire was administered both pre- and post-VR exposure, versus administration only after VR exposure. Specifically, post-VR scores for the nausea subscale were approximately two-thirds higher when participants had completed a pre-VR questionnaire. Thus, we ensured that participants completed only a single SSQ following VR exposure to minimise potential demand characteristics.

As well as subjective symptoms, cybersickness may also entail physiological changes, such as increased heart rate, respiration, tachygastric power, and changes in EEG spectral bands (Dennison, Wisti, & D'Zmura, 2016; Mazloumi Gavgani, Nesbitt, Blackmore, & Nalivaiko, 2017; Kim, Kim, Kim, & Ko, 2005). While these physiological measures are not currently widely employed, they can provide a more unbiased measure of cybersickness than questionnaires, avoiding potential issues of demand characteristics (Young et al., 2007). Accordingly, in Chapter 7 we measured participants' heart rate before, during, and after VR exposure, in addition to the SSQ and FMS, thus providing a physiological correlate to the subjective symptoms.

Psychophysics

In Chapter 4, we investigated participants' sensitivity to vestibular signals following exposure to vection in VR. Perceptual sensitivity may be assessed simply by calculating the percentage of trials in which the participant successfully identifies the presence of the stimulus (Kingdom & Prins, 2010). However, methods based on calculating the percentage correct cannot account for biases in participant responses. For instance, the percentage of correctly identified stimuli may be higher when the participant has a propensity to respond "yes" in all trials, irrespective of actual

sensitivity to the incoming sensory information (Kingdom & Prins, 2010). Accordingly, one method of assessing sensitivity while accounting for response biases is use of Signal Detection Theory (SDT) (Macmillan & Creelman, 1991; Stanislaw & Todorov, 1999).

SDT is a framework for understanding how observers can detect signals from random noise[1] (Macmillan & Creelman, 1991; Stanislaw & Todorov, 1999). In a typical experiment, observers are presented with trials containing a signal, such as a weak auditory tone or faint visual cue, and trials containing no signal, and are asked to report whether or not the signal was present. Given random variation in sensory systems, trials in which no signal is present are termed 'Noise' (N) trials, while trials in which signals are present are termed 'Signal+Noise' (S+N) trials, as signals are embedded within the background noise (Macmillan, 2001). The response to each of these trial types across repeated presentations is not uniform, but can randomly vary (for example, due to fluctuations of activity within sensory systems, variations in participant attention etc.) resulting in distributions of N and S+N (Figure 1). The distributions are assumed to be gaussian, and differ only in terms of their means (Macmillan & Creelman, 1991; Stanislaw & Todorov, 1999).

The overlap between N and S+N distributions can indicate the sensitivity of the observer to the stimuli. For example, distributions which overlap extensively make discrimination between signals and noise harder, resulting in lower sensitivity to the signal. By contrast, distributions with minimal overlap suggest that the observer is able to distinguish signals from noise more easily and is thus more sensitive. The distance

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[1] SDT has also been applied to discrimination between two signals, rather than the detection of signals in noise. However, here I focus solely on detection tasks, given the underlying principles of both cases are the same.

between the means of the N and S+N distributions is given by d', and thus indicates observers' sensitivity to the incoming stimulus (Figure 1).

Observer responses in a "yes/no" detection task depend on a decision variable, the criterion (C), above which "yes" responses are generated, and below which "no" responses are generated. This results in four possible outcomes: hits (P("yes"|Signal)), misses (P("no"|Signal)), false alarms (P("yes"|Noise)), and correct rejections (P("no"|Noise)) (Figure 1). The location of C accordingly indicates the observer's response bias: a criterion shifted leftwards towards the N distribution indicates a liberal response, resulting in greater numbers of Hits and False Alarms, while a criterion shifted rightwards towards the S+N distribution indicates a conservative bias, resulting in more Correct Rejections and Misses.

Calculation of both d' and C depends on the observer responses. The proportion of hits (H) and false alarms (FA) are converted into z scores (z(H) and z(FA) respectively), such that proportions above 0.5 result in positive z scores and proportions below 0.5 result in negative z scores, while an exact proportion of 0.5 results in a z score of 0. d' is then given by the difference between z(H) and z(FA):

$$d' = z(H) - z(FA)$$

Higher d' therefore reflects greater sensitivity to the signal, while a d' of 0 reflects an inability to discriminate between signal and noise (i.e., H = FA).

While d' depends on the difference between H and FA, C depends on their sum, reflecting the propensity of the participant to respond "yes" throughout the experiment. C therefore calculated as:

$$C = \frac{-(z(H) + z(FA))}{2}$$

A C value of 0 reflects equal FA and Miss (M) rates, while negative values indicate FA>M and positive values indicate M>FA. Negative C values thus indicate a liberal criterion, whereby the participant is more likely to respond "yes" than "no", while positive C values indicate a conservative criterion, with the participant more likely to respond "no" than "yes".
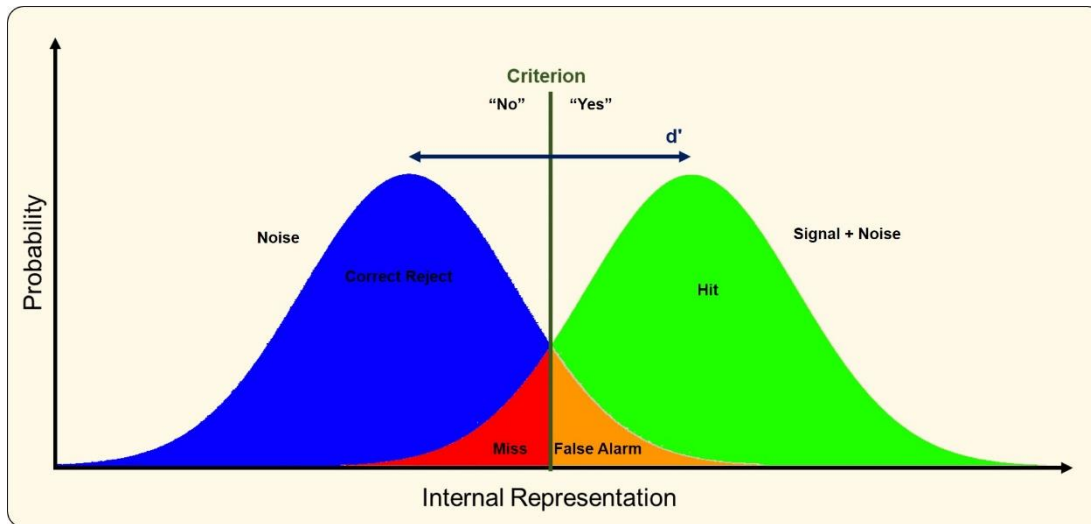
Figure 1. Decision Space for Signal Detection Theory. Noise only trials (left) and Signal+Noise trials (right) result in gaussian distributions. The separation between the two distributions indicates the participants' sensitivity to the Signal (d'), while the criterion (C) determines the participants' response bias. Hits occur when the participant responds "yes" when the signal is actually present (green). Misses occur when the participant responds "no" when the signal is present (red). False alarms occur when the participant responds "yes" and the signal is absent (orange). Correct rejects occur when the participant responds "no" and the signal is absent (blue).

In Chapter 4, we therefore devised a vestibular signal detection task, in which participants had to respond "yes" if they felt illusory motion from GVS and "no" if they did not. GVS and Sham trials were interspersed, allowing us to calculate hits (responding "yes" with GVS present), misses (responding "no" with GVS present), false alarms (responding "yes" with GVS absent) and correct rejections (responding "no" when GVS absent). Perceptual sensitivity was given by d', while response bias

was calculated as the criterion (C), with both calculated from hits and false alarms according to the equations above. Accordingly, we found that participants' sensitivity was affected by exposure to plane-congruent vection in VR, while response bias was unaffected by VR exposure.

While the vestibular detection task allowed us to assess the sensitivity of participants to incoming vestibular stimuli, it did not provide information about the participants' experience of the GVS-induced illusory motion itself. Thus, to explore the correspondence between the GVS-induced illusory motion and natural motion we used an adaptive psychophysical procedure. Detailed procedures are provided in Chapter 8, but briefly, participants were asked to indicate whether they perceived a roll rotation to the left or right while receiving concurrent natural vestibular stimulation and GVS in opposite directions. Different velocities of natural stimulation were used in order to find the velocity at which the illusory motion was cancelled by the physical motion, identifying the natural equivalent of GVS. Data were fitted with cumulative normal psychometric functions, providing the point of subjective equality (PSE, i.e., the velocity in degrees at which GVS and natural rotation sensations were cancelled) and slope (which indicated the participants' precision). Accordingly, we found that the illusory motion at 1 mA and 2.5 mA GVS was equivalent to a roll rotation towards the cathode of approximately 2°/s and 6°/s respectively, while precision was decreased with higher amplitudes of stimulation.

The QUEST+ adaptive psychophysical protocol (Watson, 2017) was used to determine the velocities of each trial. This procedure is based on Bayes' Theorem, where a prior of the psychometric function parameters is specified and combined with a likelihood function. The likelihood function is calculated from the participants' responses to estimate the posterior probability distribution of the threshold (Kingdom

& Prins, 2010; Watson, 2017). The best fitting threshold value is thus used as the stimulus intensity for the subsequent trial (Kingdom & Prins, 2010; Watson, 2017). Like other adaptive psychophysical protocols, the QUEST+ adaptive protocol can therefore obtain robust estimates of the PSE and slope faster than non-adaptive procedures. Given that each trial is selected on the basis of previous responses, it may be possible that trial dependencies begin to emerge (Kingdom & Prins, 2010). However, to reduce this problem, we interleaved two staircases corresponding to the polarity of GVS, such that participants could not easily predict the direction of rotation on the basis of the previous trial.

Conclusion

To explore the visuo-vestibular multisensory integration framework for cybersickness, a range of multidisciplinary research methods from vestibular research, VR, and psychophysics were combined. These techniques included natural and artificial vestibular stimulation, measurement of vestibular reflexes, VR optic flow and virtual environments, cybersickness measures, Signal Detection Theory and adaptive psychophysical protocols. Thus, the combination of these methods allowed a thorough exploration of vestibular processing after VR exposure, as well as clear assessments of new methods for cybersickness reduction. Importantly, we used a hypothesis-driven approach, with well-established techniques to address our research questions.

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Chapter 3:

Cybersickness: A multisensory integration perspective

Gallagher, M., & Ferrè, E. R. (2018). Cybersickness: A Multisensory Integration Perspective. *Multisensory Research*, *31*(7), 645-674. doi:10.1163/22134808-20181293

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Summary

In the past decade, there has been a rapid advance in Virtual Reality (VR) technology. Key to the user's VR experience are multimodal interactions involving all senses. The human brain must integrate real-time vision, hearing, vestibular and proprioceptive inputs to produce the compelling and captivating feeling of immersion in a VR environment. A serious problem with VR is that users may develop symptoms similar to motion sickness, a malady called cybersickness. At present the underlying cause of cybersickness is not yet fully understood. Cybersickness may be due to a discrepancy between the sensory signals which provide information about the body's orientation and motion: in many VR applications, optic flow elicits an illusory sensation of motion which tells users that they are moving in a certain direction with certain acceleration. However, since users are not actually moving, their proprioceptive and vestibular organs provide no cues of self-motion. These conflicting signals may lead to sensory discrepancies and eventually cybersickness. Here we review the current literature to develop a conceptual scheme for understanding the neural mechanisms of cybersickness. We discuss an approach to cybersickness based on sensory cue integration, focusing on the dynamic re-weighting of visual and vestibular signals for self-motion.

Introduction

Virtual Reality (VR) came to the public's attention in the late 1980s. However, the real growth in popularity of VR has been observed in more recent years, when the development in technology allowed a user to be immersed in a completely virtual world through the use of 3D real-time computer graphics and advanced display devices. A report by Goldman Sachs in January 2016 predicted that revenue from VR would approach $80 billion by 2025, with 315 million users (Bellini et al. 2016). Pivotal to the user's VR experience are multimodal interactions involving all senses. The human brain must integrate real-time vision, hearing, vestibular, and proprioceptive inputs to produce a compelling and captivating feeling of immersion and presence in a VR environment akin to real life scenarios (Azmandian et al. 2016; Herbelin et al. 2015). VR has proved to be beneficial in several applications ranging from educational and training platforms, to recreational gaming or media viewing, as well as flight simulators and medical rehabilitation (Alaker et al. 2016; Pelargos et al. 2016; Valmaggia et al. 2016; Viñas-Diz and Sobrido-Prieto 2016).

A troublesome problem with VR is that between 20% and 80% of users exhibit symptoms that parallel symptoms of classical motion sickness (Cobb et al. 1999; Munafo et al. 2017). This so-called cybersickness can be profoundly unsettling, and may compromise well-being and performance in VR training (Cobb et al. 1999; Sharples et al. 2008; Fiore et al. 2013; Llorach et al. 2014; Munafo et al. 2017). Symptoms of cybersickness include discomfort, apathy, nausea, drowsiness, disorientation, eyestrain and fatigue (Stanney et al. 1997; Rebenitsch and Owen 2016). Although its causes are not yet entirely clear, cybersickness occurrence might be dependent on different factors, including the user's gender (Munafo et al. 2017), age (Arns and Cerney 2005), duration of VR exposure (Liu 2014), and hardware issues,

such as lag (i.e., delays in updating the visual scene following a user's movement or action) or flicker (i.e., flashing of the visual scene relating to refresh rates) (Moss et al. 2011).

In the past decade, there has been a rapid advance in VR to increase the simulation realism by means of integrated multimodal experiences (i.e. 3D audio, haptic and force feedback, etc.). Significant technical improvements have also occurred, including better positional tracking, display resolution, and display refresh rate (Harsora et al. 2017). For example, many Head Mounted Displays (HMDs) (i.e., Oculus Rift CV1 and HTC Vive) are capable of tracking the position of the head in space via numerous sensors and cameras, have a refresh rate of 90Hz, and visual resolution of 1080x1200 resolution for each eye. The improvement in VR technology is therefore self-evident, and even more if compared to earlier versions of commercial HMDs (i.e., no positional tracking capabilities, a 60Hz refresh rate, and resolution of 640x800 for each eye) (Harsora et al. 2017). Moreover, the earliest HMDs had much more limited technical capabilities: 30 frames per second, a 40 degree field of view, and rather cumbersome head position sensors (Sutherland 1968). Further improvements are likely in the coming years, such as foveated rendering and adaptive displays which increase the resolution and refresh rate of the display at selected locations of the VR scene (Padmanaban et al. 2017; Lee et al. 2017; Hsu et al. 2017), increasing realism and immersion in VR (see Bastug et al. 2017 for a review). Despite these advances, there are still some issues in VR and as one leading iconoclast in VR technology - Palmer Luckey - has indicated, "VR isn't perfect right now" (Kushner 2016). Cybersickness remains a barrier to VR use: it has even been argued that the more realistic the VR environment, the more likely it is that the user experiences cybersickness (Merhi et al. 2007; Davis et al. 2015). Although the exact reason behind

this is still unclear, better resolution of optic flow might potentially exacerbate visual-vestibular-proprioceptive conflicts (Kennedy et al., 2003; Merhi et al., 2007; Davis, Nesbitt and Nalivaiko, 2015). Alternatively, one can hypothesise that the increased feeling of presence (i.e. the feeling of "being there" in a VR environment) due to the availability of detailed simulated environments might exacerbate cybersickness symptoms (Kennedy et al., 2003; Merhi et al. 2007; Davis et al. 2015). In such cases, it is possible that the detailed, realistic environment and increased presence may render the conflict in sensory signals more dramatic, increasing cybersickness. Further research is necessary to explore the exact relationship between increased feeling of presence and cybersickness.

The root causes of cybersickness remain poorly understood, however more general accounts of motion sickness have been adapted as explanations given the similarity between cybersickness and other types of motion sickness. These theories include Reason and Brand's (1975) Neural Mismatch Theory, based on sensory conflict, and Riccio and Stoffregen's (1991) Postural Instability Theory based on movement control. However, neither theory can fully explain why cybersickness arises, nor can they predict an individual's likelihood of developing symptoms. Despite these theories highlighting the role of multiple sensory inputs in the cause of cybersickness, more recent advances in knowledge of multisensory integration have only recently applied to this field (Balter et al. 2004; Oman 2012; Jürgens et al. 2016; Weech and Troje 2017).

Here we review the current literature to provide an overview of cybersickness and perceptual after-effects induced by VR, as well as evaluating the current theories of cybersickness. We highlight how dynamic re-weighting of sensory cues can be implicated in cybersickness development and after-effects of VR exposure. This

approach may provide insight into cybersickness, facilitating further research and leading to developments which may enhance the VR experience.

The vestibular system and sensory integration for self-motion perception

In everyday life, our perception of self-motion depends on the coherent integration of visual, proprioceptive and vestibular information. When we move in the surrounding environment, the visual system provides retinal-image motion (optic flow) cues, the proprioceptive receptors in the muscles, tendons, and joints sense the relative position of body parts in space and the vestibular system encodes angular and linear acceleration.

Since Gibson in 1950, motion related retinal-images have been considered essential for eliciting sensations of self-motion displacement (Gibson, 1950). However, it is now recognised that extra-visual cues make an equal contribution to self-motion perception. In the absence of visual cues, humans rely on vestibular information to estimate bodily motion (Israël and Berthoz 1989; Irsaël et al. 1993; Berthoz et al. 1995). The vestibular system is a set of sensory organs located in the inner ear, comprising of three orthogonal semicircular canals (anterior, posterior and horizontal) that sense rotational acceleration of the head in three-dimensional space and around three cardinal axes (yaw, roll, pitch), and two otolith organs (utricle and saccule) that code translational acceleration, including the orientation of the head relative to gravity. Vestibular organs are extremely sensitive to even the slightest changes in rotation and linear movement of the head, providing the feedback necessary for the brain to dictate adjustments that allow the body to maintain balance. Dynamic vestibular inputs from the semicircular canals are associated with low-level visuo-

vestibular interactions to control gaze and eye fixation, while gravitational inputs from the otolith organs contribute to path integration and navigation. Thus, vestibular cues are fundamental for perception of self-motion, aiding us in distinguishing self from object motion and providing us with a sense of where we are in space (Green and Angelaki 2010; Greenlee et al. 2016). Thus, it might not be surprising that the vestibular system plays a vital contribution in the development of motion sickness, including cybersickness. Accordingly, labyrinthine-defective patients do not experience any motion sickness symptoms (Cheung et al. 1991; Paillard et al. 2013) while blind individuals do, suggesting that visual information, while implicated motion sickness, is not as crucial as vestibular signalling (Greybeil 1970).

Multimodal interactions between visual, somatosensory, proprioceptive and vestibular signals have been described in almost all vestibular relays, including the vestibular nuclei, the thalamus and several areas in the cerebral cortex (Lopez et al. 2012; Zu Eulenburg et al. 2012). Electrophysiological studies have identified a widespread vestibular network in which the core area is the Parieto-Insular Vestibular Cortex (PIVC) (Guldin and Grüsser 1998; Chen et al. 2010). This area consists of the posterior insula/retroinsular cortex in the bank of the lateral sulcus (Guldin and Grüsser 1998). The human homologue of the primate PIVC is a distributed set of regions, including retroinsular cortex, temporoparietal junction and somatosensory cortices (Fasold et al. 2002; Lopez and Blanke 2011; Zu Eulenburg et al. 2012). Neuroimaging studies with artificial vestibular stimulation showed activation in large swathes of the cortex, including activations of classically unimodal sensory and motor regions, supporting function integration between signals and inputs from other senses (Ferrè and Haggard 2015).

Multisensory interactions between sensory modalities are fundamental in shaping our perceptual experiences. Sensory signals presented simultaneously in more than one sensory channel tend to be detected more accurately than the same signals presented individually (Stein et al. 1996). Most studies of vestibular-multisensory interactions have focused on multisensory convergence between vestibular and visual signals. Multisensory convergence involves multisensory integration of different signals related to a common external source object or percept (Ernst and Bülthoff 2004). Critically, the neural mechanism underlying multisensory convergence is likely to be a process seeking to reduce perceptual uncertainty about the source (Knill and Pouget 2004), which often involves optimal combination of cues across modalities (Ernst and Banks 2002).

The predominant theme in recent electrophysiological work has been the convergence between vestibular signals and visual signals for perception of self-motion, spatial orientation, and navigation in the environment. Multisensory neurons coding for visual and vestibular signals have been described in the macaque ventral intraparietal area (VIP, Bremmer et al. 2002), which is considered homologous to human vestibular areas in the posterior parietal cortex (Lopez and Blanke 2011). Visuo-vestibular interactions are often interpreted within the framework of optimal cue combination for multisensory perception of a single underlying quality (Gu et al. 2008; Fetsch et al. 2009). For instance, heading direction is accurately perceived in visuo-vestibular multimodal conditions. Macaques trained to complete a heading discrimination task where cues were provided by an optic flow (vision), or by a motion platform (vestibular), or combined (visuo-vestibular), showed smaller thresholds for detecting head direction under the combined sensory condition than either of the unimodal visual or vestibular conditions (Gu et al. 2008). Importantly, these results

were mirrored by the activity of dorsal medial superior temporal (MSTd) neurons: the neurons' preferred heading direction was similar for both visual and vestibular modalities, suggesting a neural mechanism for perceptual integration. Fetsch et al. (2009) further investigated this integration by dynamically modifying the reliability of the visual cue by reducing optic flow coherence and placing the visual and vestibular cues in conflict. The results matched the predictions of optimal cue integration; as the coherence of the visual cue decreased and became less reliable, the weighting of the vestibular cue increased. Interestingly, the conflict between the visual and vestibular cues did not prevent sensory integration. Similar results were found in human participants. Thus, visual and vestibular signals might be combined optimally when estimating heading direction – and therefore self-motion (however see de Winkel et al. 2010; de Winkel et al. 2013 for contrary findings). The activity of neurons in MSTd is a likely neural mechanism for this effect (Angelaki et al. 2011).

What is cybersickness?

Cybersickness is an unpleasant sensation, comprising of symptoms of disorientation, drowsiness, eyestrain, and nausea arising from exposure to immersive VR environments. Cybersickness is triggered by visually-induced illusory motion within an immersive VR environment, in which an optic flow provides motion information in the absence of corresponding vestibular signals (Reason and Brand 1975; Hill and Howarth, 2000; Keshavarz et al. 2015; Rebenitsch and Owen 2016). For this reason, cybersickness is slightly different compared to traditional motion sickness syndromes, such as car-sickness, sea-sickness and air-sickness, in which the physical movement of the vehicle triggers motion sickness symptoms (Reason and

Brand, 1975; Golding 2016). However, given its similarity to motion sickness, cybersickness can be regarded as a type of visually induced motion sickness.

Many VR applications employ an optic flow pattern which elicits an illusory feeling of self-motion, namely vection. In a VR driving simulator, for instance, the simulation provides accurate optic flow patterns of the road, buildings and other parts of the environment, eliciting clear vection sensations. The visual signals tell the user that they are moving in a certain direction with a certain acceleration. However, since the user is not actually moving, the vestibular organs provide no cues for linear or angular acceleration. As visual signals for self-motion are not corroborated by inertial forces transmitted through the vestibular system, a sensory conflict is likely to occur, and subsequently lead to cybersickness (Keshavarz et al. 2015).

The example above refers to the scenario in which the user does not move their head during VR exposure. However, many VR HMDs are now supplied with positional trackers, which enable the user to physically move in the real world while exploring the VR environment (Harsora et al. 2017). When physically moving in VR, visual cues are supported by vestibular information. This may drastically reduce the conflict between sensory modalities, and may prevent the occurrence of cybersickness. At present, few empirical studies have compared levels of cybersickness between locomotion techniques, and findings are somewhat mixed (Chance et al. 1998; Zanbaka et al. 2004; Peck et al. 2011; Llorach et al. 2014). For example, Llorach et al. (2014) compared navigation in a VR environment via a game controller, in which the user remained stationary and moved via controlling joysticks, and a position estimation system which tracked the users' actual movements. Levels of cybersickness were much higher when the user did not move and used the game controller compared to when they navigated by physically moving. By contrast, Peck et al. (2011) found no

differences in cybersickness symptoms between VR scenarios in which users freely moved, walked in place, or explored the VR environment using a joystick, despite finding better performance in the free movement scenario. Although new developments in positional trackers also allow users to explore the VR environment through head movements while sitting, vection appears much stronger when users move their heads, and possibly contributing to cybersickness (Ash et al. 2011). In addition, it is not possible to exclude that incorrect updating of the visual scene during active head motion might elicit cybersickness (Ash and Palmisano 2012). For example, cybersickness increases when the visual VR scene moves in the same direction of the physical head movement (Palmisano et al. 2017). Similar conflicts between visual and vestibular signals are implicated in car-, sea-, and space-sickness (Lackner 2014).

Traditionally, the presence of cybersickness has been explored via subjective self-reports. The Simulator Sickness Questionnaire (SSQ; Kennedy et al. 1993) is the most frequently used. This questionnaire breaks down motion sickness symptoms into three main categories: disorientation (D), including symptoms such as dizziness, vertigo and difficulty focusing, oculomotor (O), including eyestrain, headache, and blurred vision, and nausea (N), including stomach awareness, increased salivation, as well as nausea itself (Table 1). Cybersickness is characterised by severe and frequent disorientation symptoms, followed by nausea symptoms, and least oculomotor symptoms (a so-called D>N>O profile; Stanney et al. 1997; Rebenitsch and Owen 2016). This symptom profile further distinguishes cybersickness from other types of motion sickness. For example, simulator sickness has an O>N>D profile, sea-sickness an N>O>D profile, and space sickness an N>D>O profile (Stanney et al. 1997; Rebenitsch and Owen, 2016). Moreover, symptoms of cybersickness are reportedly

much more severe than simulator sickness and other motion sickness symptoms (Kennedy et al. 2003).

Table 1. Symptoms and Physiological Changes in Cybersickness

Symptoms of cybersickness falling under each category of Kennedy et al.'s (1993) Simulator Sickness Questionnaire, and reported physiological changes caused by cybersickness.

Symptoms of cybersickness (according to the Simulator Sickness Questionnaire Categories)			Physiological changes in cybersickness	
Nausea	*Oculomotor*	*Disorientation*	*Increases in*	*Decreases in*
Discomfort	Discomfort	Difficulty	Heart rate	Photoplethysmogram
Increased	Fatigue	Focusing	Respiration rate	Skin temperature
Salivation	Headache	Nausea	Skin conductance	Heart period
Sweating	Eyestrain	Fullness of	Gastric activity	EEG Theta power
Nausea	Difficulty	head	Blinks	
Difficulty	Focusing	Blurred vision	EEG Alpha power	
Concentrating	Difficulty	Dizziness	EEG Beta power	
Stomach	Concentrating	Vertigo	EEG Gamma power	
Awareness	Blurred			
Burping	Vision			

Along with self-reported symptoms of cybersickness, a range of physiological changes have also been described, including increases in tachygastric power, heart rate and eyeblinks, decreases in bradygastric power, and changes in skin temperature and EEG power bands (Kim et al. 2005; Nalivaiko et al. 2015) (Table 1). Kim et al. (2001) identified increases in eyeblinks, skin conductance response, and heart rate, as well as decreases in the amplitude of photoplethysmogram associated with cybersickness. In addition, a selective modulation of the EEG gamma band activity was noted (Kim et al. 2001). Critically, physiological changes can predict cybersickness severity. For instance, bradygastric power, breathing rate, pulse amplitude and blinking rate can

predict total or subscale scores of the SSQ (Dennison et al. 2016). Although physiological changes could represent an objective measure of cybersickness, they are not widely employed at present. Exploration of these changes may prove useful in determining the likelihood of cybersickness severity in individual users, therefore allowing intervention before cybersickness develops. An overview of cybersickness symptoms and physiological changes can be seen in Table 1.

The severity of cybersickness symptoms seems to be proportional to the duration of VR exposure: increasing exposure time within a single VR session is likely to increase cybersickness symptoms (Stanney et al. 2002; Moss et al. 2011). Liu (2014) found that participants completing a VR task had more severe symptoms as the duration increased from 5 to 15 minutes. Interestingly, a 20-minute session produced less severe symptoms than the 15-minute session, which might be due to adaptation to the VR environment (see below).

It is also possible that cybersickness symptoms do not recede immediately after cessation of VR, but rather linger on for some time following exposure. Stanney and Kennedy (1998) found cybersickness symptoms present at least one hour post-exposure. In accordance with the general profile of cybersickness, disorientation symptoms were most severe, followed by nausea and oculomotor symptoms. Startlingly, disorientation symptoms were almost 150 times higher immediately following VR exposure, and remained 95 times higher than the pre-exposure level one hour following exposure.

Although the profile of symptoms in cybersickness is relatively consistent, several factors influence whether an individual will develop the syndrome during VR exposure. Not surprisingly, people who have a history of motion sickness

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susceptibility are more likely to experience cybersickness, and are less likely to enjoy using the technology (Nichols 2000; Rebenitsch and Owen 2014). Age and gender also seem to be important: Arns and Cerney (2005) found that symptom severity and incidence increased with age, and a number of studies report that females are more susceptible to cybersickness than males (Stanney et al. 1999; Stanney et al. 2003; Flanagan et al. 2005; Kim et al. 2008; Chen et al. 2015). The reason for this gender difference is somewhat unclear, and might be driven by the influence of the menstrual cycle (Clemes and Howarth 2005; however, Golding et al. 2005 argue that the influence of the menstrual cycle is too small to fully account for the gender difference), differences in postural stability between men and women (Koslucher et al. 2016), or a larger field of view in women (LaViola 2000).

Current interpretations of cybersickness

While an understanding of cybersickness and its influences is growing, uncovering precise neural mechanisms behind cybersickness is less straightforward. A specific framework for cybersickness has not been widely employed at present. Instead, general theories of motion sickness have been adopted to explain cybersickness (Table 2).

Table 2. Theories of Cybersickness

A brief overview of motion sickness theories commonly applied to cybersickness.

Theory	Authors	Key aspects
Sensory conflict theories	Reason & Brand 1975	Sensory signals which do not match stored sensory signals generate a mismatch signal, triggering motion sickness
	Oman 1988	Sensory conflicts increase a mismatch vector, triggering motion sickness
	Bles et al. 1998	Conflicts between the sensed and predicted gravitational verticals trigger motion sickness
Other theories	Riccio & Stoffregen 1991	Postural instability causes motion sickness

Reason and Brand (1975) suggested that a discrepancy between sensory modalities is the root cause of motion sickness syndromes. Two main forms of sensory conflict have been identified in motion sickness: intersensory conflicts between visual and vestibular signals, and intrasensory conflicts between the semicircular canals and otoliths within the vestibular system (Reason 1978). Thus, the vestibular system seems to be critical in causing motion sickness, as supported by evidence in peripheral vestibular patients who do not experience any form of motion sickness (Cheung et al. 1991; Paillard et al. 2013).

According to Reason and Brand's (1975) Neural Mismatch Theory, a copy of a self-generated movement is paired with the resulting sensory inputs to form a predicted pattern of sensory cues, i.e. an engram (Reason, 1978). Then, a comparator module matches the actual sensory inputs with the stored engrams. If the input and engram do not match, a discrepancy arises and a mismatch signal is generated,

triggering motion sickness. The strength of this mismatch signal is dependent on how many sensory modalities are in conflict, the extent of the discrepancy and the amount of previous exposure to the conflicting stimuli. Accordingly, the strength of the mismatch signal corresponds to the latency and severity of motion sickness symptoms. When VR users are immersed in applications where they perceive self-motion through vection, visual signals suggesting movement conflict with vestibular inputs signalling the user is stationary. A mismatch signal is then generated if no matching engram is found, triggering cybersickness (Reason and Brand 1975; Reason 1978).

Reason (1978) proposed a further classification of sensory conflicts which have the potential to trigger cybersickness. First, information signalled by visual and vestibular systems is contradictory. For example, this may be the case when HMDs are improperly calibrated, showing VR movements (visual cues) that are not properly aligned with the user's head movements (vestibular cues). Second, visual information is not corroborated by expected signals from the vestibular organs. This sensory conflict is unsurprisingly common in cybersickness, when vection is not supported by vestibular information. Finally, vestibular information is not corroborated by visual signals, as experienced in the use of HMDs without head-tracking in which changes in head position may not be verified by changes in the VR scene.

Although Reason and Brand's (1975) theory is widely accepted, it cannot fully account for motion sickness, and therefore cybersickness, onset and development. First, this framework lacks a clear physiological basis which would explain the importance of mismatch signals in facilitating sickness (Oman 1988). Second, Reason and Brand's (1975) theory cannot account for individual differences in motion sickness (Warwick-Evans et al. 1995; Davis et al. 2014). For example, it is unclear why females should be more susceptible to motion sickness than males (Stanney et al.

1999; Stanney et al. 2003; Flanagan et al. 2005; Kim et al. 2008; Chen et al. 2015). Finally, the theory is unable to explain why some sensory cues are more likely to cause sickness than others. According to Neural Mismatch Theory, any sensory conflict triggering a mismatch signal should cause sickness, so it is unclear why particular stimuli are more nauseogenic than others. For example, scene oscillations within a VR environment are more likely to cause cybersickness than scenes with no oscillation (So and Lo, 1999; Lo and So, 2001). In particular, oscillations of around 0.2Hz in real motion sickness are highly nauseogenic, with oscillation along the fore-aft axis the most likely to cause sickness (Kennedy et al. 2010).

To address some of these open questions, Oman (1988) proposed that a desired body state prompts muscle activity and postural changes to reach that state. These changes provide signals which, along with external noise, are detected by different sensory modalities. An internal model based on all sensory modalities is formed, which is compared with actual sensory signals, providing a difference vector. Accordingly, greater sensory conflicts lead to a larger vector, which may reflect severe sickness.

Building upon the theory of Oman (1988), Bles et al. (1998) and Bos et al. (2008) proposed a more nuanced description of sensory conflict based on perception of the subjective vertical. The subjective vertical is formed from integrated sensory information from vision, proprioception, and the vestibular organs and is necessary for successful interactions with the external world (Barra et al. 2010). The visual and vestibular senses construct a model of the expected subjective vertical, as well as sensing the actual subjective vertical. The comparison between the sensed and expected verticals leads to a difference vector, prompting motion sickness. Motion sickness may therefore arise when there is an unexpected change in the subjective vertical, causing a conflict between the sensed and expected verticals. In the case of

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cybersickness, the VR environment may contain aspects where the visual and vestibular vertical are at odds, however further research is necessary to unpack aspects of this theory of motion sickness.

While the underlying basis for sensory conflict-based models remains debated, empirical evidence for the involvement of sensory conflicts in motion sickness induced by physical movement (i.e., sea-sickness, car-sickness) highlights their contribution to the development of sickness symptoms (Kato and Kitazaki 2008; Wang and Lewis 2016; Wada et al. 2016; Wada and Yoshida 2016). The specific evidence for visuo-vestibular conflicts in cybersickness is growing, suggesting that the strength of the sensory conflict between visual and vestibular cues can lead to increased sickness (Bonato et al. 2009; Nishiike et al. 2013). For example, Akiduki et al. (2003) induced a visuo-vestibular conflict while participants were immersed in VR. Participants were instructed to follow a virtual ball around the room, allowing for a range of movements. The visuo-vestibular conflict was induced by doubling the range of movement of the VR environment background relative to the participants' head movements. Cybersickness symptoms were significantly greater during and immediately after VR exposure. In addition, more complex patterns of visual motion are also related to cybersickness. Keshavarz and Hecht (2011) found that rotation across two or three axes induced increased levels of sickness than a single axis of rotation. Thus, greater mismatches between visual and vestibular modalities might trigger symptoms of cybersickness, as purported by Reason and Brand (1975) and Oman (1988).

According to the above theories, sensory conflicts are the cause of motion sickness, however it is unclear how and why such conflicts would cause symptoms such as nausea. One well-cited hypothesis is that of Poison Theory (Treisman 1977). According to this hypothesis, sensory mismatches are part of an early warning system

when an animal has ingested toxins. Nausea is therefore an adaptive, evolved response to sensory conflict aimed at ridding the animal of dangerous toxins (Treisman 1977). While this hypothesis is a plausible explanation for nausea symptoms of motion sickness, it does not account for other symptoms, such as oculomotor or disorientation symptoms, and many authors argue that it is not a compelling explanation for motion sickness (LaViola 2000; Oman 2012; Davis et al. 2015).

Riccio and Stoffregen (1991) proposed that motion sickness is a result of prolonged postural instability: people are likely to suffer motion sickness when experiencing novel situations for which they have not yet learned strategies to stabilise their posture (Stoffregen et al. 2000; Villard et al. 2008). For example, Stoffregen and Smart (1998) found increases in postural sway preceding symptoms of visually induced motion sickness when participants were exposed to low-amplitude optical flow in an immersive environment. Moreover, Smart et al. (2002) found that pitch velocity and vertical variability could predict which participants would become sick when exposed to optic flow stimulation. However, the causal relation between postural instability and cybersickness is not yet clear. For instance, Dennison and D'Zmura (2017) found that postural sway was similar both before and during VR exposure, and cybersickness increased both when participants were seated (and therefore unlikely to have an unstable posture) and when they were standing (and thus subject to greater postural demands and the potential for instability). Similarly, Warwick-Evans et al. (1995) found that motion sickness was equally present when participants viewed a video while standing and while restrained in a chair. Finally, Akiduki et al. (2003) demonstrated that postural instability (in particular, body sway) was only significantly different post-exposure to VR, pointing to instability as a consequence, rather than a cause, of cybersickness.

Is it possible to prevent cybersickness?

Techniques for preventing cybersickness include adaptation to VR through repeated exposure (Barrett 2004; Keshavarz 2013), designing VR environments to include stable visual references of the horizon or perceptual vertical (Han et al. 2011), developing applications based on physical locomotion (Llorach et al. 2014), and providing concurrent vestibular signals by means of galvanic vestibular stimulation (Cevette et al. 2012).

Adaptation to VR is arguably a more readily available technique to prevent cybersickness, as it requires the user to repeatedly engage with VR, rather than modification of VR applications or use of sensory substitution equipment (Keshavarz 2013; Golding 2016). Several studies have shown reduced cybersickness following adaptation to VR: participants exposed several times to the same VR scenario showed decreased cybersickness symptoms (Regan 1995). Similarly, Hill and Howarth (2000) asked participants to complete five sessions across five days in which they played a racing game for 20 minutes via a HMD. Some participants also passively viewed the scene while the experimenter played the game, increasing their exposure to VR. Participants experienced at least a mild degree of malaise during the first session, however by the end of the fifth session seven out of 11 participants who played and watched the game reported no cybersickness symptoms, suggesting faster adaptation to VR. These findings are in accordance with the mentioned theories of motion sickness. For example, Reason and Brand's (1975) Neural Mismatch theory predicts that as participants are further exposed to conflicting sensory stimuli, their neural store creates a new engram, and in subsequent exposures a mismatch signal is not generated to trigger cybersickness. Similarly, an internal model of expected sensory signals within the VR environment may be updated as participants are further exposed to the

conflicting stimuli, as predicted by Oman (1988) and Bles et al. (1998). By contrast, Riccio and Stoffregen's (1991) postural instability hypothesis predicts that individuals adapt to motion sickness stimuli when they learn new strategies to control their posture in the provocative environment. While adaptation to a cybersickness-inducing environment is possible, it is yet unclear which of these motion sickness theories best describes the process by which adaptation might occur.

Although the beneficial effects of VR adaptation on cybersickness symptoms are promising (Howarth and Hodder 2008; Moss et al. 2011), several drawbacks are apparent. First, repeated exposures are necessary for adaptation to be effective, which implies significant commitment from VR users. Second, it is not yet clear how durable the benefits of adaptation may be, limiting its utility. Finally, an inverse relation exists between reduction of cybersickness through adaptation and the development of VR after-effects. These include altered visual perceptions and balance problems (Stanney and Kennedy 1998; Stanney et al. 1999; Di Girolamo et al., 2001; Harm et al. 2008). Thus, the more habituated an individual becomes to the virtual world, the more likely they are maladapted to the real world on VR cessation, incurring a range of after-effects (Wright 2014).

Sensory conflict appears the most likely explanation of cybersickness. Thus, a reduction of these conflicts might prevent cybersickness. In VR the conflicting information between vestibular and visual cues prevents the user from being able to accurately assess self-motion. The presentation of 'rest frames' has been proposed as a method for reducing cybersickness (LaViola 2000; Han et al. 2011). Rest frames are an explicit frame of reference for spatial information concerning stationary objects, providing information on which to base self-motion priors. For instance, cybersickness scores were reduced when users were exposed to a VR rollercoaster scenario with a

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superimposed grid compared to a standard VR scene (Chang et al. 2013), as Bles et al.'s (1998) subjective vertical conflict hypothesis would predict: by providing a clear frame of reference for the visual vertical in VR, conflicts between predicted and experienced subjective verticals are minimised.

Recent VR scenarios allow users to physically move in the VR environment, reducing the conflict between visual and vestibular systems (Llorach et al. 2014). Despite the possibility of cybersickness reduction through physical locomotion, limitations of physical space, particularly for home users of VR, may mean that users prefer to remain stationary and navigate the virtual environment by other means (for example, controllers) (Williams et al. 2007; Riecke et al. 2010). In addition, errors in position tracking and lag, while significantly improved in more recent HMDs, may also contribute to cybersickness development (Fiore et al. 2013; Kinsella et al. 2016; Palmisano et al. 2017).

Sensory conflicts can also be reduced by matching visual cues with artificial vestibular signals. Cevette et al. (2012) applied artificial vestibular stimulation (Galvanic Vestibular Stimulation) while participants used a flight simulator. Galvanic Vestibular Stimulation induced illusory sensations of self-motion which were purported to match the visual signals experienced by the participants. Since the conflict between visual and vestibular signals was reduced, a significant reduction in sickness symptoms was found. Similarly, Galvanic Vestibular Stimulation applied during turns in a driving simulator was suggested to reduce scores in motion sickness questionnaires and improve performance (Reed-Jones et al. 2007). In addition, Galvez-Garcia et al. (2015) found that applying Galvanic Cutaneous Stimulation either continuously or intermittently while participants used a driving simulator reduced sickness scores relative to a condition with no stimulation. These results suggest that

the use of artificial stimulation may be a potential method for preventing cybersickness in VR.

VR after-effects: A re-adaptation to the real world

After-effects can arise following exposure to a variety of different sensory stimuli. One interesting and well-studied after-effect following exposure to passive motion, such as on a sea voyage, is so-called mal de debarquement. This syndrome induces illusory feelings of self-motion, such as bobbing or swaying lasting for days or even years (Van Ombergen et al. 2016). Although less severe, after-effects can frequently develop in the hours and days following a prolonged VR experience (Kellog et al. 1980; Gower and Fowlkes 1989). In one of the most bizarre cases, a pilot had his view of the world invert 180 degrees while driving a car hours after having been in a VR flight simulator (Kennedy et al. 1987). As a result of these after-effects, many air force bases have mandatory polices which stipulate that pilots cannot fly an aircraft up to 24 hours after exposure to a VR simulator. Also, many VR entertainment centres require that users do not drive for several minutes after exposure. It is important to note that some VR applications are designed specifically to carry over sensorimotor or behavioural changes following exposure to the stimulus, for example for rehabilitation or training purposes (Cameirao et al. 2011; Verschure 2011; Cameirao et al. 2012; Alaker et al. 2016; Pelargos et al. 2016; Valmaggia et al. 2016; Viñas-Diz and Sobrido-Prieto 2016). However, for the purposes of this review we focus only on maladaptive after-effects.

After-effects might be induced by adaptation to conflicting sensory stimuli. One of the most well-known examples is adaptation to prism lenses (Redding et al.

2005). On first wearing the lenses, which displace the visual field, participants make frequent errors in pointing and grasping. However, participants quickly adapt to the lenses and their accuracy increases. On removal of the glasses, however, participants begin to make errors and a period of re-adaptation is necessary before performance returns to normal (Clower et al. 1996). Adaptation to VR may follow a similar pattern, however a thorough exploration of after-effects of VR exposure has not yet been conducted.

Harm et al. (2008) found that proprioceptive coordination between the eyes, head and hands was worse following 20 minutes of VR exposure. Critically, the performance was not only immediately worse, but approached recovery only by 6 hours post-exposure. Similarly, participants showed increased pointing errors immediately after 30 minutes of VR exposure (Stanney et al. 1999).

Oculomotor after-effects have also been described after adaptation to VR. Di Girolamo et al. (2001) found that vestibular-ocular reflex gain decreased immediately following 20 minutes of VR exposure, and took 30 minutes to return to baseline levels. Although preliminary, these results provide an insight into the potential complications of VR exposure.

A multisensory integration perspective for cybersickness

The evidence reviewed above suggests a pervasive influence of multisensory interaction in VR experience. In this section, we outline a conceptual scheme of how these could underlie cybersickness. Sensory inputs constantly reach the human brain. However, these signals need to be integrated to provide successful descriptions of the environment. Vestibular inputs make an essential contribution in this process,

assessing whether visual signals are consistent or not with the movement and position of our head in space. Critically, when self-motion signals provided by the vestibular system cannot be aligned with those from visual cues, multisensory conflict occurs, potentially triggering cybersickness.

Under normal conditions, the visual and vestibular systems interact to provide information about self-motion. Optimal multisensory integration involves higher weighting of more reliable signals (Ernst and Banks, 2002). Thus, sensory cues are weighted according to their reliability, such that vestibular cues are given higher weighting when visual cues are unreliable and vice versa. A growing body of research has shown that visuo-vestibular integration is near-optimal for self-motion, with an overweighting of the vestibular cue relative to "true" optimality. Several results suggest that a process based on dynamic sensory reweighting may be important to explain cybersickness and adaptation to virtual environments (Butler et al. 2010; de Winkel et al. 2010; Fetsch et al. 2010; Angelaki et al. 2011). Three results are particularly relevant here. First, the contribution of optic flow to perceived self-motion typically emerges only after several seconds of exposure to visual cues when inertial cues are not present, implying that a dynamic process of sensory re-weighting is necessary to resolve sensory conflicts before the perception of self-motion emerges (Young et al. 1973). Second, when vestibular and visual signals are conflicting, the optimal combination of cues cannot occur without a re-weighting of the original cues. In the real world, the vestibular cue is overweighted relative to optimality (Kaliuzhna et al. 2016, but see de Winkel et al. 2010). This implies that the sensory conflict experienced in virtual reality must be resolved by substantial down-weighting of vestibular cues and up-weighting of visual cues. Third, the results on visual or vestibular dominance in such conflicting conditions appear to vary across studies,

suggesting that the human brain dynamically chooses which sensory cues are relevant for a particular situation (Young et al. 1973; Zacharias and Young 1981; Probst et al. 1985). However, taken together these results suggest that dynamic reweighting may be implicated in cybersickness.

Cybersickness may be due to the consequences of a conflict between the sensory signals which provide information about the body's orientation and motion. Visual signals tell users that they are moving in the environment. Since users are not actually moving, their vestibular architecture provides no corresponding cues of linear and angular acceleration. In view of this, when the perception of visual self-motion is not supported by inertial forces transmitted through the vestibular organs, a visuo-vestibular conflict is likely to occur, leading to cybersickness. Alteration of the weight of vestibular cues provides a means of resolving the visuo-vestibular conflict, reducing symptoms of cybersickness.

Multisensory theories suggest that multiple sensory signals need to be combined, and that the nervous system faces a key challenge in selecting the correct weighting for each signal in the combination. As in other cases of sensory conflict, resolution occurs by weighting each individual sensory signal according to its importance. In VR, the brain tends to habituate to extract self-motion information from visual cues in a visuo-vestibular conflicting environment. Since vestibular information is usually highly reliable in determining the body's position and motion in space (Prsa et al. 2012; Kaliuzhna et al. 2016), the vestibular signals must be down-weighted to avoid the occurrence of sensory conflicts. We suggest that a dynamic re-weighting function is an important element of cybersickness which has not yet been extensively researched. In VR, the dynamic re-weighting function increases the weight of visual signals about motion and decreases the weight of vestibular information responsible

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for self-motion. As a result, the visuo-vestibular conflict is decreased and therefore no longer perceived, reducing cybersickness. Cybersickness symptoms typically develop within the first minutes of VR exposure (Stanney and Kennedy 1998; Davis et al. 2015). Thus, the re-weighting function must rapidly respond to the visuo-vestibular conflict to prevent the occurrence of symptoms. If the re-weighting function is slow to respond to the conflict, cybersickness may ensue. Interestingly, decreasing the reliability of vestibular cues by applying artificial noisy Galvanic Vestibular Stimulation has been shown to modulate vection perception (Weech and Troje 2017). This might be due to rapid re-weighting of visual and vestibular information. Critically, this approach can be implemented as a method to reduce cybersickness, as proposed by Weech and Troje (2017).

On return to the real world the individual is likely to move and explore the environment, causing a flow of both vestibular and visual signals about self-motion. After exposure to VR, the brain tends to habituate to extract self-motion information from visual cues in a vestibular-conflicting VR environment. As for traditional sensory adaptation phenomena, a form of negative correlation between a current percept and the adapted stimuli may take place (Barlow and Hill 1963). An error correction process needs to occur in order to de-correlate the current percept from the adapted stimuli. Thus, a further re-weighting of both sensory signals is necessary for the optimal integration of sensory input. A period of re-adaptation must occur, whereby the vestibular cue must be up-weighted and visual cue down-weighted. During this period of re-adaptation, VR after-effects may occur over time until the vestibular cue is weighted to its usual state. The time-course of these after-effects is not yet fully known and may range from a period of minutes to hours or days (Kellog et al. 1980; Gower and Fowlkes 1989; Harm et al. 2008).

Traditionally, cybersickness has been explained by prior knowledge of predicted sensory consequences of self-motion (Reason and Brand 1975). Our conceptual scheme suggests a dynamic on-line re-weighting function of sensory cues for self-motion which can explain cybersickness, adaptation, and after-effects of VR. Reciprocal inhibitory vestibular-visual interactions support this hypothesis. Accordingly, PET studies using artificial vestibular stimulation demonstrated not only an activation of the PIVC but also a decrease in rCBF of the visual cortex (Wenzel et al. 1996; Brandt et al. 1998; Deutschländer et al. 2002). Similarly, Bense et al. (2001) showed deactivation of the occipital visual cortex induced by vestibular stimulation, and deactivation of the vestibular areas during optic flow.

Suggestions for future research

The conceptual scheme proposed here makes clear testable predictions about multisensory interactions in VR exposure and cybersickness. These could be investigated in lab-based cognitive experiments or in more applied VR scenarios. First, if the vestibular system plays a fundamental role in cybersickness, one might predict that synchronised passive movements or artificial vestibular stimulation may reduce the conflict between visual and vestibular cues, preventing sickness and forgoing the need for sensory re-weighting function in order to adapt to the VR environment. Indeed, recent reports (Reed-Jones et al. 2007; Cevette et al. 2012; Galvez-Garcia et al. 2015) have demonstrated that both galvanic vestibular stimulation and galvanic cutaneous stimulation can reduce sickness in simulators.

Second, if vestibular cues are down-weighted during VR exposure, one might expect that physiological vestibular functioning is altered during or immediately after

VR. Although this aspect has not yet been investigated, one can imagine a testing battery investigating both vestibular sensitivity and physiological functioning in order to identify whether the vestibular organs adapt to VR.

Finally, individual variability in the re-weighting function may correlate with cybersickness susceptibility. Those whose weighting function rapidly changes reliance on the vestibular cue under VR conditions are potentially less likely to experience cybersickness, as the cue conflict is rapidly resolved. By contrast, those with a higher reliance on the vestibular cue under normal conditions may be more susceptible to cybersickness as the magnitude of the cue conflict is greater. Extensive research on these predictions has yet to be conducted, however we note that Balter et al. (2004) found no difference on sensory re-weighting abilities between participants susceptible and non-susceptible to car-sickness. Participants susceptible and non-susceptible to car-sickness were repeatedly administered with galvanic vestibular stimulation while their body sway was measured. Although it was predicted that participants who were less susceptible to car-sickness would habituate to the vestibular stimulation (and therefore show reduced body sway) faster than participants who suffered from car-sickness, both susceptible and non-susceptible participants showed similar habituation gains to the vestibular stimulation. However, it is important to highlight that these findings do not refer to conflicting stimuli; it is therefore possible that susceptible and non-susceptible individuals may show differences in re-weighting when exposed to visual–vestibular conflicting signals.

Conclusion

While significant advances have been made in understanding cybersickness, there is broad scope for further advancement in this field. The symptom profile of cybersickness has been clearly delineated, as well as identification of factors which influence its development. However, explanations for why cybersickness occurs based on sensory conflict theory are tentative, and there is no identified predictive mechanism. In addition, a conspicuous gap in our knowledge concerns the after-effects of virtual reality exposure. A few studies have identified cybersickness symptoms, proprioceptive disruptions, postural instability and oculomotor symptoms as potential after-effects, however their time-course is unknown, despite the time-course of cybersickness itself being well-defined (Kennedy et al. 2000). The current sensory conflict models also cannot fully account for why these after-effects arise.

Here we argued that an approach based on multisensory integration could provide a predictive explanation for cybersickness, adaptation to VR and its after-effects. Under normal circumstances, visual and vestibular cues are optimally integrated for self-motion perception.

However, in VR these cues conflict with one another, prompting cybersickness, and must be re-weighted with higher reliance on the visual cue to allow integration. This dynamic re-weighting function has the potential to explain adaptation and after-effects of VR, as well as individual differences in cybersickness susceptibility. It is hoped that this approach could further our knowledge of cybersickness, as well as lead to clearer avenues for prevention of cybersickness symptoms.

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Chapter 4:

Multisensory Interactions in Virtual Reality: Vection reduces vestibular sensitivity, but only for congruent planes of motion

Word count: 6,150

Abstract

During exposure to Virtual Reality (VR) a sensory conflict may be present, whereby the visual system signals that the user is moving in a certain direction with a certain acceleration, while the vestibular system signals that the user is stationary. In order to reduce this conflict, the brain may down-weight vestibular signals, which may in turn affect vestibular contributions to self-motion perception. Here we investigated whether vestibular perceptual sensitivity is affected by VR exposure. Participants' ability to detect artificial vestibular inputs was measured during vection-inducing or non-vection optic flow stimuli on a VR head-mounted display. Sensitivity to vestibular signals was significantly reduced when vection-inducing stimuli were presented, but importantly this was only the case when both visual and vestibular cues conveyed information on the same plane of self-motion. Our results suggest that the brain dynamically adjusts the weight given to incoming sensory cues for self-motion in VR, however this is dependent on the congruency of visual and vestibular cues.

Key words: Vestibular System, Multisensory Integration, Self-Motion Perception; Virtual Reality.

Introduction

Moving through the world elicits a host of sensory information. Images moving across the retina provide an optic flow, while linear acceleration and angular rotation signals are detected via the vestibular organs in the inner ear. Typically, when moving through the external environment visual and vestibular inputs are perfectly matching and therefore the brain integrates them to form a coherent percept of the direction and speed of self-motion (Gu, Angelaki and DeAngelis, 2008; Fetsch et al., 2009; Butler et al., 2010; Greenlee *et al.*, 2016). According to Bayesian optimal integration accounts, multisensory integration reduces uncertainty and noise regarding the source percept (Ernst and Banks, 2002; Knill and Pouget, 2004). As such, more reliable cues are given a higher weighting than unreliable ones, and consequently bimodal sensory estimates are more precise than estimates obtained from a single sensory modality (Ernst and Banks, 2002; Ernst and Bülthoff, 2004). Evidence suggests that visuo-vestibular integration for self-motion follows exactly this Bayesian optimal integration framework: estimates of self-motion tend to be more precise when both visual and vestibular cues are available (Gu, Angelaki and DeAngelis, 2008; Angelaki, Gu and DeAngelis, 2011). Importantly, the weight given to vestibular cues increases as the coherence of the visual cues decreases (Fetsch et al., 2009).

Multisensory neurons coding for visual motion, namely *vection*, and vestibular motion were found in the macaque Middle Temporal (MT) complex: neurons in the dorsal Medial Superior Temporal area (MSTd), a subregion of this complex, strongly respond to retinal motion associated with optic flow (Tanaka and Saito, 1989; Duffy and Wurtz, 1991) and to vestibular stimulation arising from actual movement (Bremmer, Kubischik, Pekel, Lappe, and Hoffmann, 1999; Fetsch et al., 2007). Vestibular neurons responding to conflicts between predicted and actual inputs from

active and passive movements have been described in the vestibular nuclei and brainstem (Carriot, Brooks and Cullen, 2013; Oman and Cullen, 2014). Neuroimaging studies have confirmed cross-modal visual and vestibular convergence of cues to self-motion in the human homologue of MT and in the cingulate sulcus visual areas (Smith, Wall and Thilo, 2012). Reciprocal visuo-vestibular interactions are fundamental for self-motion (Brandt et al., 1998). Positron Emission Tomography (PET) studies using artificial vestibular stimulation demonstrated not only an activation of the cortical vestibular network but also a decrease in regional Cerebral Blood Flow (rCBF) of the visual cortex (Wenzel et al., 1996; Deutschländer et al., 2002). Similarly, Bense et al. (2001) showed bilateral deactivation of the occipital visual cortex induced by artificial vestibular stimulation, suggesting a neural basis for visuo-vestibular integration for self-motion.

However, there are some circumstances, such as Virtual Reality (VR), in which visual and vestibular cues for self-motion may not be available and even potentially in conflict (Reason and Brand, 1975; Bos, Bles and Groen, 2008). This is the case when VR users feel the sensation of travelling through a virtual environment, while actually remaining stationary in the real world. Consider a typical VR scenario in which the user is driving a car while actually sitting on a chair: optic flow signals that the user is moving in a certain direction with a certain acceleration, however as the user is not physically moving, the vestibular organs signal that the user is stationary. This visuo-vestibular sensory conflict seems to be the underlying mechanism for the frequently experienced *cybersickness*, a form of motion sickness induced by exposure to VR (Kennedy, Drexler, and Kennedy, 2010; Keshavarz, Hecht, and Lawson, 2014; Rebenitsch and Owen, 2016; Stanney, Kennedy, and Drexler, 1997). As such, understanding how visuo-vestibular integration for self-motion occurs in VR may

provide further insights to prevent cybersickness, and potentially improve the VR user experience.

According to Bayesian optimal integration frameworks (Ernst and Banks, 2002; Ernst and Bülthoff, 2004; Gu, Angelaki and DeAngelis, 2008; Angelaki, Gu and DeAngelis, 2011), when exposed to an environment in which visual cues are present and vestibular cues are uncertain or conflicting, such as VR, the weighting of the vestibular cues may be decreased, and the brain extracts self-motion information primarily from visual signals (Gallagher and Ferrè, 2018; Gallagher, Dowsett and Ferrè, 2019). In other words, the brain adapts to extract self-motion information from visual cues and disregard vestibular signals. This dynamic re-weighting process reduces visuo-vestibular conflict in VR, and eventually cybersickness. Accordingly, Weech, Moon and Troje (2018) demonstrated that noisy artificial vestibular stimulation reduced the reliability of vestibular information in VR, decreasing symptoms of cybersickness. Similarly, Bos (2015) reported reduced motion sickness when vibration was applied to the head to decrease vestibular reliability, suggesting that a sensory re-weighting may be implicated in different forms of motion sickness.

Critically, the ability to perceive self-motion by an optic flow may be altered by concomitant vestibular inputs (Edwards, O'Mahonys, Ibbotson, and Kohlhagen, 2010, Shirai and Ichihara, 2012, Holten and MacNeilage, 2018). The detection of optic flow stimuli was reduced when participants viewed an expanding optic flow stimulus coupled with incongruent backwards physical motion, compared to congruent visuo-vestibular conditions, i.e., expanding optic flow with forward physical motion (Edwards et al., 2010). However, findings are still somewhat mixed and evidence appears contrasting. Recent studies, indeed, reported a better detection of optic flow induced self-motion in incongruent visuo-vestibular conditions (Shirai and Ichihara,

2012), or even no differences between congruent or incongruent visuo-vestibular signalling (Holten and MacNeilage, 2018).

While vestibular input seems to modulate the perception of optic flow, it is not yet clear whether vection may affect vestibular processing. Importantly, the dynamic re-weighting process described above clearly predicts a reduction in vestibular perceptual sensitivity during exposure to VR applications which generate visuo-vestibular conflicts. It has been shown that adaptation to vection leads to motion after-effects which bias vestibular processing such that a greater physical motion is required to cancel the perceived illusory motion induced by optic flow (Cuturi and MacNeilage, 2014). Additionally, a decrease in the gain of vestibulo-ocular reflexes (VOR) has been reported after exposure to VR (Di Girolamo et al., 2001). Specifically, around 20 minutes of VR exposure dramatically decreased VOR gain by approximately 41% (Di Girolamo et al., 2001). Here we investigated whether exposure to vection induced by full field VR optic flow affects participants' sensitivity to vestibular input. In Experiment 1, we administered low intensity, short-duration Galvanic Vestibular Stimulation (GVS) while participants viewed patterns of rotating dots inducing roll vection, or randomly moving dots, inducing no vection. Binaural GVS delivered between the mastoids activates the peripheral vestibular organs, i.e., the otoliths and semicircular canal afferents (Stephan et al., 2005; Cullen, 2019; Kwan et al., 2019), producing a polarity-dependent *virtual roll-rotation vector* (Fitzpatrick and Day, 2004; Cathers, Day and Fitzpatrick, 2005). GVS-induced self-motion percepts are polarity dependent: left-anodal and right-cathodal GVS mimics an inhibition of the left and an activation of the right ear vestibular peripheral organs, decreasing the firing rate of the vestibular nerve on the left side and increasing it on the right side which is perceived as a movement towards the right (Goldberg, Smith and Fernández, 1984). In contrast,

right-anodal and left-cathodal GVS induces the opposite effect. We hypothesised a reduction in perceptual sensitivity to vestibular input while viewing vection versus no-vection stimuli. Further, we investigated whether the presence of vection itself may be enough to modulate vestibular sensitivity or whether visual and vestibular cues for self-motion must be congruent in order to interact. In Experiment 2, we therefore explored whether the modulation of vestibular sensitivity is *generally* induced to the presence of vection, or whether it is *specifically* caused by the congruency of visual and vestibular cues for self-motion.

Experiment 1: Congruent visuo-vestibular cues for self-motion in VR

Methods

Ethics

The experimental protocol was approved by the ethics committee at Royal Holloway, University of London. The experiment was conducted in line with the Declaration of Helsinki. Written informed consent was obtained prior to commencing the experiment.

Participants

Twenty-four naïve participants (8 male, age M = 20.71, SD = 2.27) completed the experiment. All participants were right-handed according to their Edinburgh Handedness Inventory (Oldfield, 1971) scores. Exclusion criteria were any history of neurological, psychiatric, or vestibular disorders, epilepsy or family history of

epilepsy. All participants had normal or corrected-to-normal vision. Data from two participants were excluded as they were more than 2 standard deviations from the mean in at least one condition, resulting in a total sample of 22 participants for analysis.

Galvanic Vestibular Stimulation (GVS)

Bipolar GVS was applied to deliver a boxcar pulse of 0.7 mA with 250 ms duration, based on our previous study (Cabolis, Steinberg and Ferrè, 2018). We used GVS parameters which induced a relatively faint virtual sensation of roll rotation. Individual thresholds for GVS induced roll-rotation sensations range from 0.4 to 1.5 mA (Kerkhoff et al., 2011; Oppenländer et al., 2015), with recent studies suggesting average thresholds of approximately 1.8 mA for short (500-2000ms) boxcar GVS pulses (Ertl, Klimek, Boegle, Stephan, and Dieterich, 2018).

Electrodes (approx. 4 cm^2) were coated with NaCl gel and affixed to each of the mastoid processes. Left-anodal/right-cathodal stimulation (L-GVS) induced a sensation of roll rotation towards the right, whereas the reverse polarity (R-GVS) induced a sensation of roll rotation towards the left. Sham stimulation was also used as a control. Two electrodes were placed on the neck, approximately 5 cm below the upper electrodes, using both left-anodal/right-cathodal stimulation (L-SHAM) and right-anodal/left-cathodal stimulation (R-SHAM). The sham stimulation controlled for cutaneous sensations experienced during GVS, as well as the knowledge that an unusual stimulation was occurring. No sensations of self-motion were experienced during this type of stimulation. GVS and sham stimulation waveforms were generated by a custom-written code in LabView (LabView 2012, National Instruments) and

conveyed to a commercial stimulator (Good Vibrations Engineering Ltd., Nobleton, ON, Canada) over serial port.

Experimental design and procedure

Data from each participant was gathered in a single session. Verbal and written instructions about the task were given to participants at the beginning of the session. Participants were asked to wear an Oculus Rift CV1 head-mounted display (HMD). To reduce the postural consequences of the GVS pulse, the experiment was conducted in a comfortable sitting position and participants were asked to rest their head in a chinrest and place their arms on the table in front of them.

Our design factorially combined vection and vestibular signals. The *Vestibular Detection Task (VDT)* was designed to follow a signal detection approach (Macmillan and Creelman 1991) (Figure 1A). Participants' sensitivity to vestibular cues was determined by asking them to detect trials on which GVS was present ("signal" trials) versus trials in which Sham stimulation was present ("noise" trials). Both sensitivity to vestibular cues and response bias could therefore be compared during exposure to vection-inducing or non-vection inducing visual motion. Thus, a 2 (vestibular stimulus present/absent) $\times$ 2 (vection stimulus present/absent) design, with the following trial types: 30 vestibular only trials (vestibular stimulus present and vection stimulus absent); 30 vestibular and vection trials (vestibular stimulus present and vection stimulus present); 30 vection only trials (vestibular stimulus absent and vection stimulus present); and 30 no stimulus trials (vestibular stimulus absent and vection stimulus absent). Thus, a total of 120 trials were performed divided into four blocks.

Half of the vestibular present trials was presented with L-GVS and the other half with R-GVS. Sham stimulation (L-SHAM and R-SHAM) was administered in the vestibular absent trials. In the vection present trials, full-field vection visual dots were presented on the Oculus HMD. Approximately 500 dots rotated anticlockwise at 90°/s, inducing a sensation of roll-vection. Crucially, this sensation of vection is congruent with the self-motion sensation induced by GVS. In the vection absent trials the dots moved randomly, inducing no sensations of self-motion. All visual trials included a fixation cross at the centre of the HMD, and participants were asked to always fixate on the fixation cross. The visual stimulus was presented for 60s prior to completing the detection task and continued throughout the entire block (total presentation of approximately 4 minutes). Vection present and absent stimuli were presented in separate blocks. Vection was described as the illusion one experiences when watching a neighbouring train move while sat stationary (Keshavarz et al., 2015). In particular, participants were told that it might feel as if they were rotating to one side or the other.

On each trial, participants heard a beep to indicate that they should pay attention to any potential GVS-induced roll sensations, but ignore any non-specific vestibular sensations, such as tingling under the electrodes surface. A second beep 500ms later indicated that participants should verbally respond "yes" if they felt roll sensations or "no" if they did not. GVS/SHAM stimulation was delivered between these two sounds. The visual stimulus remained on the HMD throughout the experimental trials. A custom LabView program was used to trigger the stimuli and record participant responses.

Data analysis

A signal detection approach was used to analyse the VDT data (Macmillan and Creelman, 1991). The number of hits (the number of trials in which L-GVS/R-GVS was present and the participant responded "yes"), misses (the number of trials in which L-GVS/R-GVS was present and the participant responded "no"), false alarms (the number of trials in which L-SHAM/R-SHAM stimulation was present and the participant responded "yes"), and correct rejections (the number of trials in which L-SHAM/R-SHAM stimulation was present and the participant responded "no") were calculated. Hit rates [P("yes"|GVS)] and false alarm rates [P("yes"|SHAM)] were used to calculate perceptual sensitivity (d'), the difference between z transformed probabilities of hits and false alarms [d' = z(Hit) – z(False Alarm)]. This therefore represented the separation between Sham ("noise") and GVS ("signal+noise") distributions according to the SDT decision framework (Macmillan and Creelman, 1991). Greater values of d' thus indicated greater sensitivity to vestibular signals, while values of d' closer to 0 indicated lower sensitivity. The response bias (C), the tendency for participants to report the GVS stimulus as present, was also calculated [C = -[z(Hit) + z(False Alarm)]/2]. C values of 0 represented equal probability of responding "yes" and "no". Negative values represented a liberal response bias, whereby participants were more likely to respond "yes", resulting in greater false alarms than misses. Positive values represented a conservative response bias, with participants more likely to respond "no", resulting in greater misses than false alarms. Both d' and C were calculated for each GVS polarity (with L-SHAM false alarm rates paired with L-GVS hit rates and R-SHAM false alarm rates paired with R-GVS hit rates) and vection condition for each participant. Data from two participants were excluded as they were above 2 standard deviations from the mean in at least one condition.

<p style="text-align: center;">**Results**</p>

Means and SDs of hits, misses, false alarms and correct rejections by vection condition and GVS polarity can be seen in Table 1, and raw values for each participant can be found in Appendix 5, Table A2.

Table 1. Mean (SD) hits, misses, false alarms, and correct rejects per vection condition and GVS polarity.

	L-GVS		R-GVS	
	No Vection	Vection	No Vection	Vection
Hits	18.95 (8.02)	14.05 (7.88)	19.41 (7.58)	13.41 (7.70)
Misses	11.05 (8.02)	15.95 (7.88)	10.59 (7.58)	16.59 (7.00)
False Alarms	3.05 (3.70)	7.95 (6.89)	4.32 (5.78)	7.36 (6.44)
Correct Rejections	26.95 (3.70)	22.05 (6.89)	25.68 (5.78)	22.64 (6.44)

Perceptual Sensitivity (d')

Raw d' values can be seen in Appendix 5, Table A1, while individual data points are plotted in Appendix 4, Figure A1. A 2x2 repeated measures ANOVA was conducted on d' values, with factors GVS Polarity (L-GVS vs. R-GVS) and Vection (Vection Present vs. Vection Absent). This analysis revealed a significant main effect of Vection ($F(1, 21) = 36.03$, $p < .001$, $\eta_p^2 = .63$) (Figure 1B). Participants' sensitivity to vestibular stimulation was significantly lower following Vection Present (M = 0.69, SD = 0.67) compared to Vection Absent (M = 1.88, SD = 1.09) trials. No significant main effect of GVS Polarity ($F(1, 21) = 0.19$, $p = .67$, $\eta_p^2 = .01$) was found. No significant interaction between Vection and GVS Polarity was found ($F(1, 21) = 0.32$, $p = .58$, $\eta_p^2 = .02$).

Response bias (C)

Raw C values can be seen in Appendix 5, Table A1, while individual data points are plotted in Appendix 4, Figure A2. A 2x2 repeated measures ANOVA conducted on C values, with factors GVS Polarity (L-GVS vs. R-GVS) and Vection (Vection Present vs. Vection Absent), revealed no significant main effects of Vection ($F(1, 21) = 0.07$, $p = .79$, $\eta_p^2 = .004$) or GVS Polarity ($F(1, 21) = 0.01$, $p = .91$, $\eta_p^2 = .001$) (Figure 1B). No significant interaction between Vection and GVS Polarity was found ($F(1, 21) = 2.31$, $p = .14$, $\eta_p^2 = .10$).

Discussion

Sensitivity to vestibular signals was significantly reduced following exposure to vection-inducing visual cues compared to randomly moving visual stimuli. Response bias was not influenced by exposure to vection in VR. Thus, our results suggest that exposure to vection in VR reduces the weighting placed on vestibular cues for self-motion. Importantly, the self-motion sensations induced by GVS and the vection inducing stimulus were congruent: both vestibular and visual cues triggered a sensation of motion on the roll axis. Thus, it is not clear whether the presence of vection itself may be enough to modulate vestibular sensitivity or whether visual and vestibular cues must be congruent in order to interact. We hypothesised that the reduction in vestibular sensitivity is selective for exposure to vection congruent with the type of movement evoked by GVS. To further investigate this hypothesis, in Experiment 2 we administered GVS during exposure to linear vection or randomly moving dots. This allowed us to explore whether the decrease in vestibular sensitivity

is *generally* due to the presence of vection, or whether it is *specifically* caused by the congruency of visual and vestibular cues for self-motion.

Experiment 2: Incongruent visuo-vestibular cues for self-motion in VR

Methods

Ethics

The experimental protocol was approved by the ethics committee at Royal Holloway, University of London. The experiment was conducted in line with the Declaration of Helsinki. Written informed consent was obtained prior to commencing the experiment.

Participants

Twenty-four naïve participants (8 male, age M = 21.63, SD = 5.13) completed the experiment. None of the participants had taken part in the previous experiment. All participants were right-handed according to their Edinburgh Handedness Inventory (Oldfield, 1971) scores. Exclusion criteria were as Experiment 1. All participants had normal or corrected-to-normal vision. Data from two participants were excluded as they were more than 2 standard deviations from the mean in at least one condition, resulting in a sample size of 22 for analysis.

Experimental design and procedure

In order to investigate whether the effects of vection on vestibular sensitivity were generic or specific to the plane of self-motion evoked by GVS (i.e. roll rotation), here the participants were administered with a full-field linear vection stimulus during vection present trials (Figure 1C). Each of the approximately 500 dots were assigned a random scaling factor between 0.01 and 1.5. On each frame, each dot expanded in size by its scaling factor in pixels from a minimum of 1 to a maximum of 9 pixels in diameter. Once the maximum size was reached, the size reset to 1-pixel diameter. The location of the dot on each frame was determined by multiplying its default X and Y coordinates by:

$$Location = \frac{Scaling\ Factor^3}{1.5} \times 1.5$$

Thus, dots nearer the centre travelled less distance than dots farther from the centre, creating an expanding pattern and inducing a sensation of linear vection. The vection stimulus was presented for 60s, and remained on screen throughout the detection task (approximately 4 minutes total presentation time). Vection was described as in Experiment 1, however participants were told that this might feel like a sensation of moving forwards through space, rather than a sensation of rotation. The experimental design and procedure were otherwise identical to Experiment 1.

Data analysis

Data were analysed as in Experiment 1. Data from two participants were excluded as they were more than 2 standard deviations from the mean in at least one condition.

<div align="center">**Results**</div>

Means and SDs of hits, misses, false alarms and correct rejects by vection condition and GVS polarity can be seen in Table 2. Raw data for each participant can be seen in Appendix 5, Table A4.

Table 2. Mean (SD) hits, misses, false alarms, and correct rejections per vection condition and GVS polarity.

	L-GVS		R-GVS	
	No Vection	Vection	No Vection	Vection
Hits	16.14 (6.47)	16.73 (6.27)	16.82 (7.90)	14.68 (7.42)
Misses	13.86 (6.47)	13.27 (6.27)	13.18 (7.90)	15.32 (7.42)
False Alarms	4.14 (4.45)	4.09 (5.04)	3.59 (4.06)	4.05 (4.84)
Correct Rejections	25.86 (4.45)	25.91 (5.04)	26.41 (4.06)	25.95 (4.84)

Perceptual Sensitivity (d')

Raw d' values can be seen in Appendix 5, Table A3, while individual data points are plotted in Appendix 4, Figure A3. A 2x2 repeated measures ANOVA was conducted on d' values, with factors GVS Polarity (L-GVS vs. R-GVS) and Vection (Vection Present vs. Vection Absent). This analysis revealed no significant main effects of Vection ($F(1, 21) = 0.21$, $p = .65$, $\eta_p^2 = .01$) or GVS Polarity ($F(1, 21) = 0.05$, $p = .82$, $\eta_p^2 = .002$) (Figure 1D). No significant interaction between Vection and GVS Polarity was found ($F(1, 21) = 3.18$, $p = .09$, $\eta_p^2 = .13$).

Response bias (C)

Raw C values can be seen in Appendix 5, Table A4, while individual data points are plotted in Appendix 4, Figure A4. A 2x2 repeated measures ANOVA was conducted on C values, with factors GVS Polarity (L-GVS vs. R-GVS) and Vection (Vection Present vs. Vection Absent), revealed no significant main effects of Vection

$(F(1, 21) = 0.12, p = .73, \eta_p^2 = .01)$ or GVS Polarity $(F(1, 21) = 1.52, p = .23, \eta_p^2 = .07)$ on response bias (Figure 1D). No significant interaction between Vection and GVS Polarity was found $(F(1, 21) = 0.55, p = .47, \eta_p^2 = .03)$.
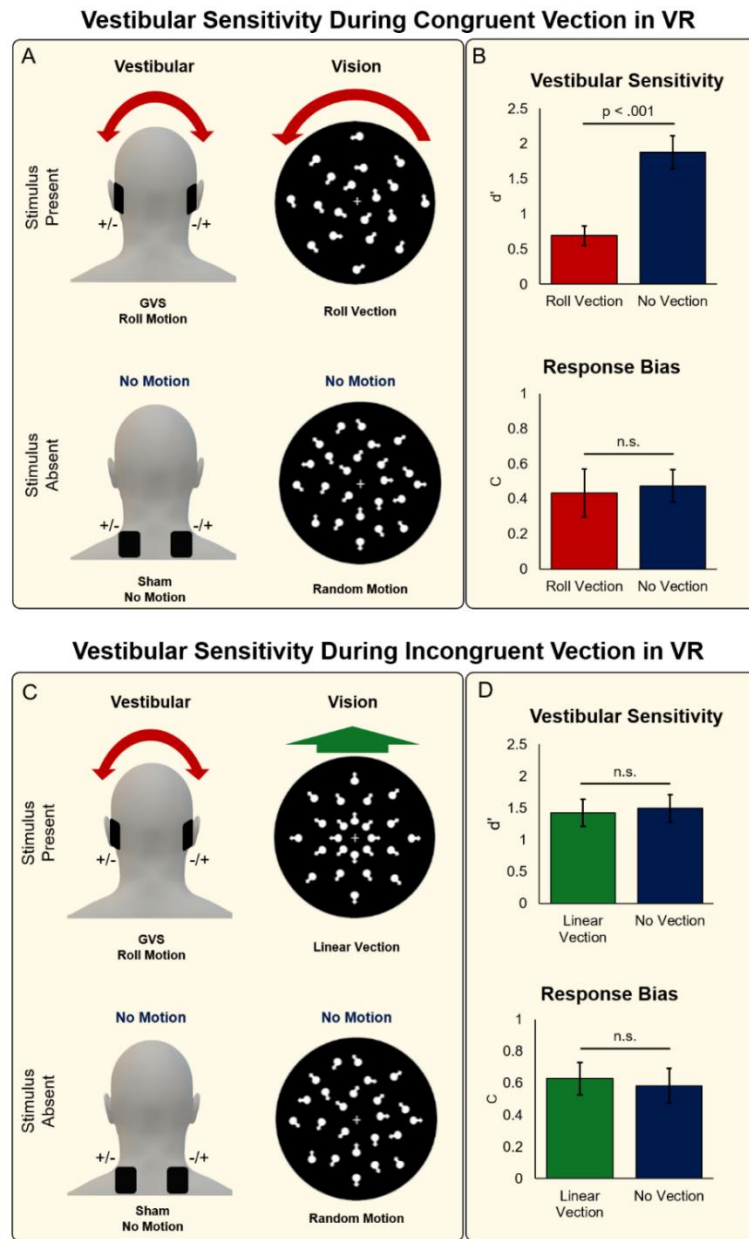
Figure 1. Experimental set up and results. A) Experiment 1 set up. Vestibular sensitivity during congruent vection in VR. We used a 2 (vestibular stimulus present/absent) x 2 (vection stimulus present/absent) design. GVS induced a sensation of roll-rotation to the left or right, while a full-field of dots in VR rotated to induce a sensation of roll vection. Sham stimulation and random motion were used as controls.B) Experiment 1 Results. Vestibular sensitivity was significantly reduced following exposure to roll vection. Response bias was unaffected by exposure to roll vection. C) Experiment 2 set up. Vestibular sensitivity during incongruent vection in

VR. We used a 2 (vestibular stimulus present/absent) x 2 (vection stimulus present/absent) design. GVS induced a sensation of roll-rotation to the left or right, while a full-field of expanding dots in VR induced a sensation of linear vection. Sham stimulation and random motion were used as controls. D) Experiment 2 Results. No changes were found on vestibular sensitivity or response bias following exposure to linear vection.

Between experiments comparisons

Our results suggest that vestibular sensitivity is reduced by vection in VR only when both visual and vestibular cues for self-motion provide information regarding the same plane of motion. To investigate this hypothesis, we directly compared the effect of congruent (Experiment 1) vs. incongruent (Experiment 2) vection on vestibular sensitivity. As no effects of polarity of GVS were found in either experiment, we averaged across L-GVS and R-GVS conditions. A *Vestibular Sensitivity Index* was estimated by subtracting the vection absent from the vection present conditions, such that positive values corresponded to greater sensitivity during vection and negative values corresponded to lower sensitivity during vection.

An independent *t*-test revealed a significant difference in the Vestibular Sensitivity Index between Visuo-Vestibular Congruent and Visuo-Vestibular Incongruent planes of motion ($t(42) = 4.44$, $p < .001$, Cohen's $d = 1.34$, 95% CI [0.69, 1.99]). Specifically, vestibular sensitivity was significantly lower during exposure to Visuo-Vestibular Congruent motion, i.e. roll vection (M = -1.19, SD = 0.93) compared to Visuo-Vestibular Incongruent motion, i.e. linear vection (M = -0.07, SD = 0.73).

Discussion

Incongruent visuo-vestibular motion signals did not influence vestibular sensitivity. Participants' sensitivity to roll rotation vestibular signals was not affected

by exposure to linear vection. However, vestibular sensitivity was significantly reduced if vection was generated on the roll plane. Thus, alterations in vestibular processing following vection in VR are dependent on the congruency between visual and vestibular cues for self-motion.

General Discussion

When moving through the world, optic flow and vestibular cues are integrated to form a coherent percept of self-motion (DeAngelis and Angelaki, 2012). During visuo-vestibular conflict, such as in VR, sensory signals may be reweighted, with more reliable sensory cues given a higher weighting (Ernst and Banks, 2002; Greenlee et al., 2016). In particular, vestibular signals may be down-weighted during VR exposure, so that the brain extracts self-motion information predominantly from visual cues (Weech and Troje, 2017; Gallagher and Ferrè, 2018). This dynamic reweighting may alter how the brain subsequently processes vestibular inputs. Here we found that participants were less able to detect vestibular signals following exposure to visuo-vestibular congruent motion in VR. Thus, changes in vestibular sensitivity occurred only when vection and vestibular sensations were congruently experienced as roll-rotation. No changes in vestibular sensitivity were found after exposure to visuo-vestibular incongruent motion. Importantly, our results indicate a specific modulation of vestibular processing induced by vection: response bias was not affected by either congruent or incongruent motion in VR. Taken together our results seem to suggest a modulation of vestibular sensitivity following exposure to vection in VR, and that this modulation depends on the specific plane of motion presented.

After-effects of VR exposure are often reported, but this remains a relatively under-explored area (Stanney and Kennedy, 1998; Gallagher and Ferrè, 2018). Altered vestibular experiences may be present in the hours or days following VR exposure (Di

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Girolamo et al., 2001; Harm et al., 2008; Stanney and Kennedy, 1998; Stanney et al., 1999). For example, disorientation scores immediately following 15 minutes of exposure to VR were 143 times higher than before VR, and remained 95 times higher 60 minutes post-exposure (Stanney and Kennedy, 1998). Sensorimotor coordination has been shown to be dramatically poorer after exposure to VR, approaching recovery only 6 hours post VR (Harm et al., 2008). Similarly, alterations in the vestibulo-ocular reflex have been reported after VR use (Di Girolamo et al., 2001). The precise causes of VR-induced after-effects are not entirely clear, however it is possible that these after-effects result from altered vestibular processing following exposure to visuo-vestibular conflict. In typical VR scenarios in which vection is used to provide a compelling sensation of self-motion, visual cues signal that the user is moving, while vestibular cues signal that they are stationary. As a result, the vestibular cues for self-motion may be down-weighted, resulting in altered vestibular processing. Here we found a decrease in vestibular perceptual sensitivity during exposure to vection in VR, but importantly, this decrease was observed only when the experienced visuo-vestibular self-motion was congruent. That is, vestibular sensitivity was poor when both visual and vestibular cues for self-motion provided information about roll-rotation, while no changes in vestibular sensitivity were found when vestibular cues signalled roll-rotation and vection provided linear acceleration sensations. Thus, our findings suggest that a dynamic reweighting of vestibular cues may impact vestibular processing during VR exposure. Future work should explore whether this dynamic re-weighting carries over after VR exposure, potentially explaining VR-induced after-effects.

Here we found decreases in vestibular sensitivity during exposure to visuo-vestibular congruent motion in VR. Previous studies have focused on the inverse

interaction, i.e., whether optic flow detection may be modulated by vestibular stimulation (Edwards et al., 2010; Shirai and Ichihara, 2012; Holten and MacNeilage, 2018). Interestingly the results are somewhat mixed. For example, Edwards et al. (2010) found that detection of optic flow was reduced when participants were exposed to incongruent vestibular stimulation. By contrast, Shirai and Ichihara (2012) found reduced detection of optic flow when it was paired with a congruent vestibular stimulus, while Holten and MacNeilage (2018) found no difference in optic flow detection between congruent and incongruent visuo-vestibular stimuli. The differences in visual and vestibular stimuli between these three studies could potentially account for these mixed findings. In particular, Edwards et al. (2010) used much faster visual stimuli and a constant acceleration vestibular stimulus, while Shirai and Ichihara (2012) and Holten and MacNeilage (2012) used slower visual stimuli and more complex vestibular motion profiles. Thus, further research is necessary to explore the relationship between stimuli types and modulation of optic flow sensitivity.

In the present experiments we investigated participants' sensitivity to vestibular cues using an approach based on Signal Detection Theory (SDT) (Macmillan and Creelman, 1991). Sensitivity to sensory cues could also be investigated by simply reporting participants' hit rates during exposure to vestibular stimulation. However, this measure does not account for biases in participant responses. For instance, participants who have a more liberal response bias are likely to respond "yes", irrespective of whether the vestibular stimulus is present or not. While this results in greater hit rates, it does not necessarily indicate greater sensitivity to vestibular cues. By contrast, the sensitivity measure used here, d', indicates participants' sensitivity independently from the response bias, C. As such, this can be regarded an appropriate method for assessing sensitivity to vestibular cues.

Subsequently, we found differences in vestibular sensitivity following exposure to congruent optic flow in the absence of changes in response bias. Interestingly, while here we focused on a detection approach, SDT could also be applied to *discrimination* of sensory cues. For instance, it may be possible to explore whether participants ability to discriminate between L-GVS and R-GVS sensations is affected by exposure to congruent or incongruent optic flow by asking them to report whether they felt roll motion to the left or right, rather than reporting the presence or absence of roll in general. Given that exposure to visuo-vestibular conflicts is predicted to result in a down-weighting of vestibular cues (Gallagher and Ferrè, 2018), one may therefore predict a decrease in discriminability of GVS types following exposure to optic flow. Finally, while the SDT approach here highlighted decreases in sensitivity to vestibular cues, it cannot highlight any phenomenological changes in the vestibular sensation induced by optic flow, for instance whether participants perceived changes in the magnitude or direction of the illusory motion induced by GVS. Further research is necessary to explore this possibility.

We investigated vestibular sensitivity during exposure to only a few minutes of vection in VR. Specifically, participants viewed the vection stimulus for 60s prior to commencing the detection task and continued viewing the visual stimuli throughout the task, resulting in approximately four minutes of visual stimulation. It is likely that the changes in vestibular sensitivity may differ according to the duration of VR exposure: for instance, sensitivity to vestibular stimuli may be higher during the first few seconds of exposure to congruent vection, declining only over time as the vestibular cue is gradually down-weighted. Interestingly, both vection and optokinetic after-nystagmus have been demonstrated to change with habituation to optic flow (Brandt, Dichgans, and Buchele, 1974). Specifically, the velocity of vection slows or

ceases with longer durations of optic flow (between 4 and 12 minutes, depending on individual variability). In addition, the amplitude of the optokinetic after-nystagmus increases up to 60s of exposure to optic flow, declining after 3 and up to 15 minutes (Brandt et al., 1974). Thus, further exploration of the time-course of vestibular sensitivity across shorter and longer periods of time will be an important step.

Curiously, while we found significant changes in vestibular sensitivity only during visuo-vestibular congruent motion, the congruency between the direction of GVS polarity and roll vection had no impact on vestibular sensitivity. This may be due to different reasons. First, GVS parameters were set in order to induce a very mild motion sensation. Thus we cannot exclude that the stimulation would have been too weak to trigger a conflict between the perceived direction of GVS motion and the direction of roll vection. Second, binaural GVS induces a polarity-dependent virtual roll-rotation vector (Fitzpatrick and Day, 2004; Cathers, Day and Fitzpatrick, 2005): left-anodal/right-cathodal GVS is perceived as a movement towards the right, while right-anodal/left-cathodal GVS is perceived as a movement towards the left (Goldberg, Smith and Fernández, 1984). However, when the stimulation is off a motion after-effect is easily perceived by participants. That is left-anodal/right-cathodal GVS generates a movement towards the right and an after-effect towards the left. It might therefore be possible that the short duration of our GVS pulses might make the direction of movements unclear. Mandatory fusion accounts might explain the decrease in vestibular sensitivity induced by congruent vection stimuli: when congruent visual and vestibular cues for self-motion are integrated, perceptual access to the unimodal estimates is lost, potentially resulting in lower sensitivity for the unimodal stimulus alone (Prsa, Gale and Blanke, 2012; Zhang et al., 2019). This account might have predicted that vestibular sensitivity would be reduced only for the

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direction-congruent polarity if mandatory fusion were the underlying mechanism. Thus, the observed modulation of vestibular sensitivity for both L-GVS and R-GVS polarities suggests a more general mechanism of down-weighting vestibular cues. However, given the previously described stimulation factors (i.e., weak stimuli and motion after-effects), further exploration of this possibility is necessary.

The integration of vestibular and visual cues for self-motion is underpinned by a complex network of brain regions. When viewing optic flow stimuli, activity is increased in MT+, Cingulate Sulcus Visual Area (CSv) and Ventral Intraparietal Area (VIP), suggesting that these regions are involved in the processing of visual cues for self-motion (Kovács, Raabe and Greenlee, 2008; Wall and Smith, 2008; Cardin and Smith, 2010). Several studies report that activity in the parieto-insular vestibular cortex (PIVC) is decreased when experiencing vection in the absence of vestibular cues (Brandt *et al.*, 1998; Kleinschmidt, 2002). However, increased activity in PIVC has also been described (Uesaki and Ashida, 2015; Kirollos, Allison and Palmisano, 2017). It is possible that differences in optic flow stimuli account for these apparently discrepant findings: while constant velocity stimuli across one axis were used in studies describing decreased PIVC activity (Brandt et al., 1998; Kleinschmidt, 2002), much more complex optic flows were used in studies reporting increased PIVC activity (Uesaki and Ashida, 2015; Kirollos, Allison and Palmisano, 2017). Thus, the effects of vection on PIVC are not yet entirely clear. Nevertheless, it seems likely that the activity in PIVC, MT+ CSv, and VIP may be implicated in the dynamic re-weighting process. Here we investigated sensitivity to vestibular stimuli during exposure to constant velocity stimuli on one axis. Thus, our findings should be extended to more complex forms of motion.

Curiously, different patterns of activity may be present during linear versus roll vection. For example, Deutschländer et al. (2004) reported increased activity in visual areas during linear vection, while roll vection led to increased activity in more parietal regions. Moreover, while both roll and linear vection decreased activity in vestibular regions, deactivation was stronger for linear vection (Deutschländer et al., 2004). In the present study, we used GVS to stimulate the vestibular system. Crucially, GVS elicits a sensation of roll motion. As such, we were not able to investigate visuo-vestibular congruent motion in the linear plane. Given the reported stronger PIVC deactivations during linear vection, we may predict that vestibular sensitivity would be reduced further if the visuo-vestibular congruent motion was in the linear versus roll plane, however more complex forms of vestibular stimulation are necessary to investigate this possibility.

The uses of VR in everyday life are becoming more apparent. While the utility of VR for training, rehabilitation, gaming, and research is clear, questions over its effect on our sensory processing remain outstanding. Previous research has documented after-effects of VR exposure, however a thorough investigation of these after-effects is lacking. Here we found that exposure to vection in VR reduced sensitivity to incoming vestibular stimulation. Crucially, this reduction in sensitivity depended on the plane of visual motion presented, with reductions following visuo-vestibular congruent, but not incongruent, motion stimuli. Our findings therefore highlight how exposure to vection in VR can modulate incoming vestibular information, and provide further insights into mechanisms of visuo-vestibular integration for self-motion perception.

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Chapter 5:

Vection in Virtual Reality Modulates Vestibular-Evoked Myogenic Potentials

Word count: 4,527

Abstract

The popularity of Virtual Reality (VR) has increased rapidly in recent years. While significant technological advancements are apparent, a troublesome problem with VR is that between 20% and 80% of users will experience unpleasant side-effects such as nausea, disorientation, blurred vision, and headaches – a malady known as Cybersickness. Cybersickness may be caused by a conflict between sensory signals for self-motion: while vision signals that the user is moving in a certain direction with certain acceleration, the vestibular organs provide no corroborating information. To resolve the sensory conflict vestibular cues may be down-weighted leading to an alteration of how the brain interprets actual vestibular information. This may account for the frequently reported after-effects of VR exposure. Here we investigated whether exposure to vection in VR modulates vestibular processing. We measured vestibular-evoked myogenic potentials (VEMPs) during brief immersion in a vection-inducing VR environment presented via head-mounted display. We found changes in VEMPs asymmetry ratio, with a substantial increase in VEMPs amplitude recorded on the left sternocleidomastoid muscle following just one minute of exposure to vection in VR. Our results suggest that exposure to vection in VR modulates vestibular processing, which may explain common after-effects of VR.

Introduction

Any organism moving through its environment receives a constant stream of sensory signals about self-motion: optic flow inputs from vision, proprioceptive information about the position of the body from muscles, joints, and tendons, and inputs for acceleration via the vestibular system. This latter seems particularly important for self-motion (Green & Angelaki, 2010). Three orthogonal semi-circular canals detect rotational movements of the head in the three-dimensional space, and two otolith organs (utricle and saccule) sense translational acceleration. Vestibular inputs are integrated with signals from other sensory modalities, such as vision, proprioception and touch (Angelaki *et al.*, 2011; Ferrè & Haggard, 2015; Alberts *et al.*, 2016; Greenlee *et al.*, 2016). Multimodal interactions have been described in almost all vestibular relays, including the vestibular nuclei, the thalamus and several areas in the cerebral cortex (Lopez *et al.,* 2012; Zu Eulenburg *et al.*, 2012). Such sensory convergence architecture reflects its key role in self-motion, and the redundancy with other modalities, described above.

Under normal circumstances, sensory signals for self-motion are successfully integrated to produce a coherent representation of the organism in the external environment. However, conflicts between sensory modalities may occur when sensory signals carry discrepant information. This seems be the case in Virtual Reality (VR). The popularity of VR has increased rapidly in recent years. While significant technological advancements are apparent, a troublesome problem with VR is that between 20% and 80% of users experience unpleasant side-effects such as nausea, disorientation, blurred vision, and headaches – a malady known as *Cybersickness* (Stanney *et al.*, 1997; Munafo *et al.*, 2017). Critically, many VR applications induce an illusory sense of self-motion, namely *vection* (Palmisano *et al.*, 2015). During

vection in VR, the user is stationary while feeling a compelling sense of translation or rotation induced by optic flow. Consider for instance a typical VR scenario, in which a VR user is driving a car. The simulation provides accurate optic flow patterns of the road, buildings and other parts of the environment. Thus, the visual signals tell the user that they are moving in a certain direction with certain acceleration. However, since the user is not actually moving, the vestibular organs signal that the user is stationary, causing a sensory conflict which may lead to VR-induced motion sickness.

The underlying mechanisms of Cybersickness are not entirely clear and several theories have been proposed (Riccio & Stoffregen, 1991; Bles *et al.*, 1998; see Rebenitsch & Owen, 2016, for a review). However, evidence suggests that Cybersickness, as in more general motion sickness (Reason & Brand, 1975; LaViola, 2000; Rebenitsch & Owen, 2016), may be triggered by visuo-vestibular conflict in VR. Accordingly, the severity of sickness symptoms increases with increased visuo-vestibular conflict (Akiduki *et al.*, 2003; Keshavarz & Hecht, 2011). It might be surprising that recent improvements in VR technology have not reduced Cybersickness: although several improvements in refresh rate, display resolution, position tracking of HMDs have increased realism in VR no significant reduction of Cybersickness has been observed (Shafer *et al.*, 2017; 2019). Moreover, VR applications with greater levels of realism have been shown to increase levels of Cybersickness, possibly due to even stronger visuo-vestibular conflicts (Stanney *et al.*, 2003; Merhi *et al.*, 2007; Davis *et al.*, 2015). Thus, VR requires the brain to adjust incoming sensory information to extract self-motion information in a vection-only environment.

Senses are usually integrated and weighted according to their reliability, with increased weight placed on more reliable ones (Stein *et al.*, 1996; Ernst & Bülthoff,

2004; Knill & Pouget, 2004). As such, when a sensory modality becomes unreliable the weighting placed on it is lowered, and other sensory modalities are given higher weighting (Ernst & Banks, 2002). Electrophysiological evidence supports this optimal integration framework for visuo-vestibular integration for self-motion (Gu *et al.*, 2008; Fetsch *et al.*, 2009, 2012; Angelaki *et al.*, 2011; DeAngelis & Angelaki, 2012). For example, heading direction is more accurately detected when both visual (vection) and vestibular (acceleration) cues are present (Gu *et al.*, 2008). Moreover, as the coherence of vection cues decreases, reliance on vestibular cues increases (Fetsch *et al.*, 2009). Thus, to deal with visuo-vestibular conflicts, such as in VR, vestibular cues may be substantially down-weighted (see Gallagher & Ferrè, 2018 for a review). Accordingly, changing the reliability of vestibular cues by noisy Galvanic Vestibular Stimulation has been shown to reduce visuo-vestibular conflict in VR (Weech & Troje, 2017). Brain regions associated with visuo-vestibular integration are likely to support this process. Vection has been shown to activate MT+, CSv, precuneus and parieto-insular vestibular cortex (PIVC) (Kovács *et al.*, 2008; Wall & Smith, 2008; Cardin & Smith, 2010; Uesaki & Ashida, 2015). Importantly, activity in PIVC decreases when people experience a sensation of vection, supporting a functional modulation of vestibular activity (Brandt *et al.*, 1998; Kleinschmidt, 2002; however see Uesaki & Ashida, 2015, for contrasting findings).

Changes in vestibular functioning may be reflected by altered vestibular experiences occurring during or in the hours and days following VR exposure (Lampton *et al.*, 1994; Stanney *et al.*, 1999; Di Girolamo et al., 2001). For example, vestibulo-ocular reflex gain decreases following VR exposure (Di Girolamo et al., 2001), and coordination between the eyes, head, and hands is poorer (Harm *et al.*, 2008). Furthermore, in one of the more bizarre cases, a pilot had his view of the world

invert 180 degrees while driving a car hours after being trained in a flight simulator (Kennedy *et al.*, 1987). This disturbing VR after-effect may be due to altered reliability of vestibular cues for orientation: on returning to the real world after VR, vestibular inputs are once again present – the user would possibly move in the environment – and the brain would need to re-weight the vestibular signals which have been attenuated during VR exposure. The vestibular cues are now given a higher weighting than during the VR exposure itself. The mentioned changes in vestibulo-ocular reflex gain is a proxy for this (Di Girolamo et al., 2001).

Here we investigated whether vestibular signals are modulated by a brief exposure to full field vection in VR. We measured reflex responses to sound-evoked vestibular stimulation (Vestibular Evoked Myogenic Potentials, VEMPs) after immersion in a VR environment eliciting an illusory sensation of linear vection. Importantly, VEMPs are a gold-standard measure for the functioning of the otolith receptors, widely used in clinical settings (Rosengren & Kingma, 2013). Loud sounds stimulate the saccule and generate a characteristic motor response in the sternocleidomastoid muscle which is functionally involved with neck flexion and head rotation. Thus, VEMPs could be taken as an indicator of wider vestibular-cortical changes elicited by the sensory conflict in VR. Previous research has found changes in VEMPs related to motion sickness susceptibility: motion sickness is positively correlated with both VEMPs amplitudes and asymmetry ratios (Tal *et al.*, 2013; Fowler *et al.*, 2014). Similarly, exposure to microgravity, which alters otolith functioning, caused changes in VEMPs asymmetry ratios (Clarke & Schönfeld, 2015). Therefore, we predict that similar changes may occur during exposure to VR-induced sensory conflict: exposure to vection in VR would modulate the amplitude, and subsequently asymmetry ratio, of VEMPs induced by sound-evoked vestibular stimulation.

Methods

Ethics

Written informed consent was obtained from participants before commencing the experiment. The experimental protocol was approved by the local ethics committee (Royal Holloway University of London) and the study was conducted in line with the Declaration of Helsinki. The authors declare no conflicts of interest.

Participants

Twenty-four healthy participants (17 female, M age = 21.13, SD = 3.90) completed the study. Twenty-one participants were right-handed according to their Edinburgh Handedness Inventory scores. Exclusion criteria were any history of neurological, psychiatric, hearing or vestibular disorders, epilepsy or family history of epilepsy. All participants' data was included in analysis, resulting in a sample size of 24 participants.

Visual stimuli

Visual stimuli were presented on an Oculus Rift DK2 head-mounted display (HMD) (Figure 2A). We note that other VR displays, such as CAVEs or surround screens, may cause similar conflicts in visuo-vestibular processing (Kennedy *et al.*, 2010). Thus, while not under direct consideration in the present study, we believe that the underlying mechanisms in these alternative VR displays may be similar to those when viewing stimuli via a HMD. Participants viewed a full field pattern of moving white dots on a black background (Figure 2B). In the random motion condition the dots moved randomly. In the vection condition the dots formed an expanding flow

pattern, causing a sensation of linear acceleration. Each dot was assigned a random scaling factor between 0.01 and 1.5. On each frame, each dot expanded in size by its scaling factor in pixels from a minimum of 1 to a maximum of 9 pixels in diameter. Once the maximum size was reached, the size reset to 1-pixel diameter. The location of the dot on each frame was determined by multiplying its default X and Y coordinates by $\frac{Scaling\ Factor^3}{1.5^3} \times 1.5$. Thus, dots nearer the centre travelled less distance than dots farther from the centre. A white fixation cross was displayed at the centre of the HMD. These parameters were used to investigate the specific effects of vection on vestibular processing. We thus eliminated other features usually present in more complex VR scenarios. Participants viewed the display for 60s before VEMPs recording was taken and continued to view the display until the VEMPs recording was completed.

VEMPs recording

VEMPs were measured according to standard procedures (Colebatch *et al.*, 1994; Fowler *et al.*, 2014; Clarke & Schönfeld, 2015) using BioMed eVEMP USB software and hardware. Electrodes were placed on the left and right sternocleidomastoid muscles in a bipolar configuration, with ground electrodes on the forehead and sternum or collar bone (Figure 2A). HDA 280 Sennheiser headphones were worn by the participants to deliver the stimuli. VEMPs were elicited via 500Hz tone burst stimuli of 7ms duration at 100dB SPL into the ear ipsilateral to the side of measurement. Muscle contraction was achieved by asking the participant to turn the head to the contralateral side and push the head down towards the floor while laying supine. The visual stimulus remained directly in front of the participant during the head movement, and participants were asked to maintain the correct posture while the

VEMPs measurements were taken. Measurements were recorded at 2000 Hz sampling frequency when the software detected that muscle tension was between 120 and 400 µV RMS and electrode impedance less than 20kΩ. Given that VEMPs amplitudes depend on the activation of the sternocleidomastoid muscle, we used a repeated measures design whereby each participant completed both vection and random motion conditions on both muscle sides. The first side of measurement was counterbalanced across participants while the visual condition was randomised within each muscle side. One hundred single trials of 80ms duration were averaged to give the final VEMP measurement, with amplitudes and latencies provided automatically by the eVEMP software. An example VEMP waveform from a representative participant can be seen in Figure 1. P1-N1 intervals were calculated by taking the time difference between N1 and P1 latencies. Asymmetry ratios were calculated accordingly, with negative values indicating higher amplitudes on the left muscle side and positive values indicating higher amplitudes on the right muscle side:

$$Asymmetry\ ratio = \frac{|P1N1Amp_r| - |P1N1Amp_l|}{|P1N1Amp_r| + |P1N1Amp_l|} * 100$$
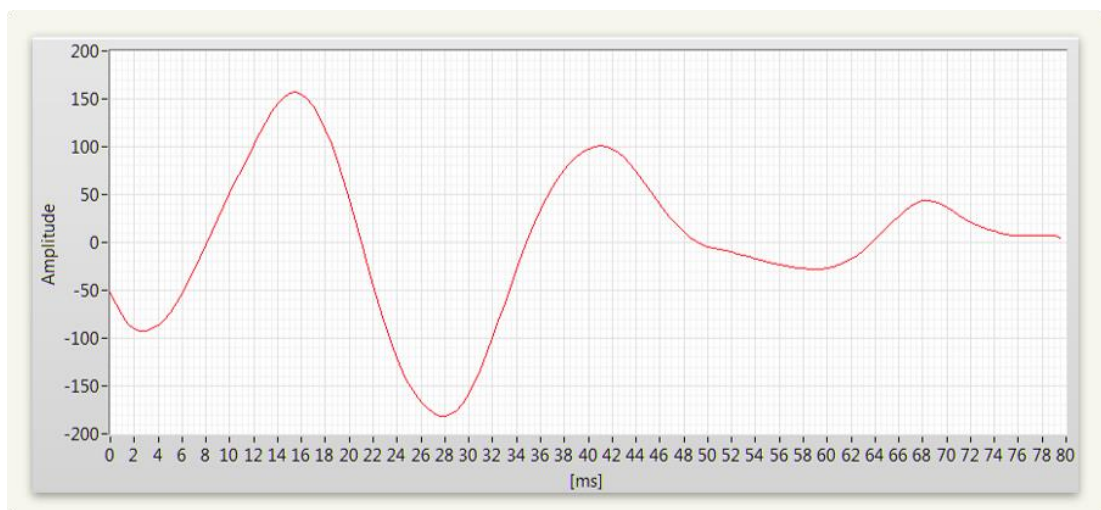
Figure 1. Example VEMP waveform from a representative participant.

Procedure

After completing informed consent procedures, participants were instructed to watch the stimuli on the HMD in a relaxed supine position for one minute before turning the head to the relevant muscle side and completing the VEMP measurement. Participants first completed practice trials on each muscle side without wearing the HMD to ensure that they adopted the correct posture and to verify accurate VEMP recording. Left and right VEMPs were then recorded in both vection and random motion conditions. The first side of measurement was counterbalanced across participants, while initial visual motion type was randomised within each muscle side. Participants were instructed to rest for three minutes in between measurements to allow the muscles to relax. If measurements were not successfully obtained, the trial was repeated. Participants were asked to report whether they experienced any sensations of self-motion while watching the stimuli. Participants also completed a Motion Sickness Susceptibility Questionnaire (MSSQ, Golding, 1998) at the start of the session.

Data analysis

Differences between random motion and vection conditions and measurement side were analysed using 2x2 repeated measures ANOVAs for P1-N1 peak-to-peak amplitudes, P1 and N1 latencies, and P1-N1 intervals. Paired *t*-tests with Bonferroni correction were used to follow up any significant main effects or interactions. A paired *t*-test was conducted on asymmetry ratios between random motion and vection conditions.

MSSQ percentile scores were calculated according to Golding (2006). Pearson correlations between amplitudes following vection exposure and MSSQ percentile scores were also conducted.

Results

All raw data are available in Appendix 5, Table A5.

Vection Reports

As expected, 22/24 participants experienced self-motion during the vection condition. 3/24 participants reported motion sensations during the random condition. 2/24 reported no sensations of self-motion at all. All participants were included in the main analysis.

Asymmetry Ratio

A significant difference in asymmetry ratio was found between vection and random motion ($t(23) = -2.14$, $p = .04$, Cohen's $d = 0.42$). Specifically, asymmetry increased following exposure to vection (mean = -4.51, SD = 14.58) compared to random (mean = 1.22, SD = 12.67) motion, with larger amplitudes on the left (mean = 281.47, SD = 80.59) versus right (mean = 255.02, SD = 64.41) muscle side. Individual data points are plotted in Appendix 4, Figure A5.

P1-N1 Peak-to-Peak Amplitude

Means and SDs for P1-N1 peak-to-peak amplitudes for each condition can be seen in Table 1. No significant main effects of visual condition ($F(1, 23) = 2.26$, p

=.15, $\eta_p^2 = 0.089$) or muscle side ($F(1, 23) = 0.75$, $p = .40$, $\eta_p^2 = 0.03$), were found on P1-N1 peak-to-peak amplitudes. However, a significant interaction between vection and muscle side was found ($F(1, 23) = 4.42$, $p = .047$, $\eta_p^2 = 0.16$). Follow-up t-tests revealed a significant increase in VEMP amplitude on the left muscle side following exposure to vection (mean = 281.47, SD = 80.59) compared to random motion (mean = 252.42, SD = 63.03) stimuli ($t(23) = 2.80$, $p = .01$, Cohen's $d = 0.40$) (Figure 2C). Individual data points are plotted in Appendix 4, Figure A6.

P1 and N1 Latency

Means and SDs for P1 and N1 latencies and P1-N1 intervals for each condition can be seen in Table 1. No significant main effect of visual condition ($F(1, 23) = 0.58$, $p = .81$, $\eta_p^2 = 0.003$) or muscle side ($F(1, 23) = 0.25$, $p = .62$, $\eta_p^2 = 0.011$) was found on P1 latency. No significant interaction was found ($F(1, 23) = 2.07$, $p = .16$, $\eta_p^2 = 0.083$). Individual P1 data points are plotted in Appendix 4, Figure A7. Similarly, no significant main effect of visual condition ($F(1, 23) = 1.17$, $p = .29$, $\eta_p^2 = 0.048$) or muscle side ($F(1, 23) = 1.51$, $p = .23$, $\eta_p^2 = 0.062$) was found on N1 latency. No significant interaction was found ($F(1, 23) = 0.38$, $p = .54$, $\eta_p^2 = 0.016$). Individual N1 data points are plotted in Appendix 4, Figure A8. Finally, no significant main effect of visual condition ($F(1, 23) = 0.72$, $p = .41$, $\eta_p^2 = 0.03$) or muscle side ($F(1, 23) = 0.17$, $p = .69$, $\eta_p^2 = 0.007$) was found on P1-N1 intervals. No significant interaction between factors emerged ($F(1, 23) = 1.47$, $p = .24$, $\eta_p^2 = 0.06$). Individual P1-N1 Interval data points are plotted in Appendix 4, Figure A9.

MSSQ Correlation

The average MSSQ percentile score was 41.80%, corresponding to moderate motion sickness susceptibility. Individuals can be classified as having low susceptibility to motion sickness with percentile scores from 0-25%, moderate susceptibility from 25-75%, and high susceptibility with scores above 75%. (Golding, 2006). Accordingly, 8 participants in the present study had low susceptibility to motion sickness, 12 had moderate susceptibility, and 4 had high susceptibility. No significant correlations were found between MSSQ percentile scores and VEMPs amplitudes after exposure to vection on either the left ($r = -0.07$, $p = .76$) or right ($r = 0.18$, $p = .39$) muscle side, thus motion sickness susceptibly does not seem to influence the VR-induced increase in VEMPs amplitude.

Table 1. Means (SDs) for P1-N1 Peak-to-Peak Amplitudes, P1 and N1 Latencies, and P1-N1 Intervals by muscle side and vection condition.

	Left		Right	
	Vection	**Random**	**Vection**	**Random**
P1-N1 Amplitude (µV)	281.47 (80.59)	252.42 (63.03)	255.02 (64.41)	255.03 (40.75)
P1 Latency (ms)	15.80 (4.19)	14.78 (3.78)	14.21 (4.71)	15.56 (4.40)
N1 Latency (ms)	27.18 (3.95)	27.69 (3.77)	26.03 (4.41)	27.40 (4.16)
P1-N1 Interval (ms)	11.38 (3.20)	12.90 (4.30)	11.82 (4.23)	11.83 (4.43)
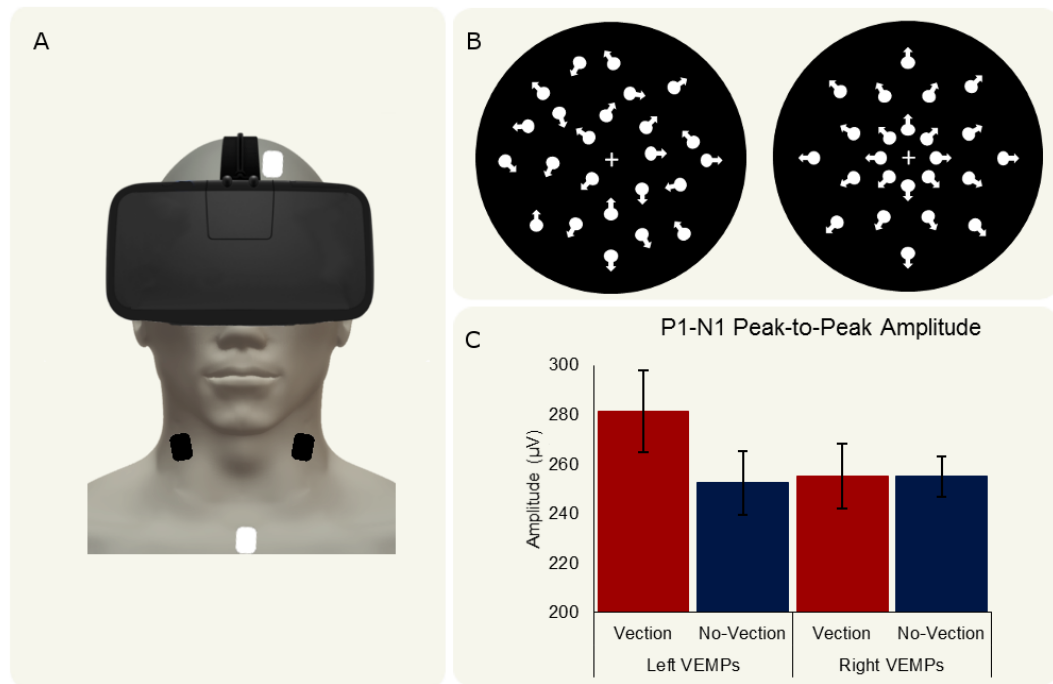
Figure 1. Experimental set up and results. A) VEMPs set-up. Electrodes were placed on the left and right sternocleidomastoid muscles (black), with ground electrodes on the forehead and sternum or collar bone (white). Headphones were worn by the participants to deliver the stimuli. VEMPs were elicited via 500Hz tone burst stimuli at 100dB into the ear ipsilateral to the side of measurement. One hundred trials were averaged to give the final VEMP measurement. P1-N1 intervals were calculated by taking the time difference between N1 and P1 latencies. B) VR stimuli. Stimuli were presented on an Oculus Rift DK2 head mounted display. Participants viewed a pattern of moving white dots on a black background. In the random condition (left) the dots moved randomly. In the vection condition (right) the dots formed an expanding flow pattern in the vection condition. The same velocity and number of dots was used in both conditions. C) VEMP amplitudes across muscle sides and motion conditions. Blue bars indicate random motion while red bars indicate vection motion. Error bars represent standard error of the mean.

Discussion

Under normal conditions, the brain optimally combines sensory signals according to their reliability (Ernst & Banks, 2002). When experiencing vection, for example in VR, the visual system signals that the user is moving through the environment (vection), however vestibular information signals that the body is stationary. This sensory conflict may subsequently lead to symptoms of Cybersickness

(Stanney & Kennedy, 1998). The brain thus has to habituate to extract self-motion information from vection in a visuo-vestibular conflicting environment (Reason & Brand, 1975; Akiduki *et al.*, 2003; Keshavarz & Hecht, 2011). To resolve this sensory conflict, vestibular signals for self-motion may be down-weighted, which may in turn affect how the brain processes incoming online vestibular information (Weech & Troje, 2017; Gallagher & Ferrè, 2018). As a result, the visuo-vestibular conflict is decreased. Critically the re-weighting must rapidly occur to counteract the occurrence of the visuo-vestibular sensory conflict. Accordingly, cybersickness symptoms have been shown to typically develop within the first minutes of VR exposure (Stanney & Kennedy, 1998; Davis *et al.*, 2014). After VR exposure, vestibular signals for self-motion are once again present and a further adjustment must occur, whereby the vestibular signals are up-weighted. Here we have found changes in vestibular processing after exposure to full field vection in VR, supporting the idea of vestibular re-weighting.

A growing body of literature suggests dynamic re-weighting of visual and vestibular cues during and after VR vection exposure. Vestibular-ocular reflex (VOR) gain is decreased immediately after VR exposure (Di Girolamo et al., 2001), and neuroimaging studies report deactivation of vestibular brain regions (i.e., PIVC) during vection (Brandt *et al.*, 1998; Kleinschmidt, 2002). These findings imply a down-weighting of vestibular cues when self-motion is experienced from vision. When vestibular cues become available, an up-weighting may occur, which may be reflected in our finding of increased left VEMPs amplitude during vection in VR. Similarly, previous studies have reported an increased reliance on vestibular cues during distance perception in VR when both visual and vestibular cues are available (Harris *et al.*, 1998, 2000; Jaekl *et al.*, 2005), and in postural control (Akizuki *et al.*,

2005) or perception of heading direction (Ter Horst *et al.*, 2015) when visual cues become unreliable. Overall, these findings therefore highlight the dynamic re-weighting of vestibular cues, which may explain adaptation and after-effects of VR exposure.

To our knowledge, no previous research has used VEMPs to investigate the effects of vection exposure on vestibular processing. VEMPs are a gold-standard measure that has been largely used both in clinical settings and research to establish the functionality of vestibular processing (Colebatch *et al.*, 1994; Rosengren & Kingma, 2013). Previous research has demonstrated alterations in VEMPs induced by motion sickness elicited by real motion, such as seasickness (Tal *et al.*, 2013; Fowler *et al.*, 2014). For instance, Fowler *et al.* (2014) showed a correlation between VEMPs amplitude and motion sickness susceptibility, with higher amplitude in individuals with high motion sickness susceptibility. We found changes in VEMPs asymmetry ratio, with a substantial increase in VEMPs amplitude recorded on the left sternocleidomastoid muscle following just one minute of exposure to vection in VR. Similarly, VEMPs asymmetry has been reported to positively correlate with susceptibility to motion sickness (Xie *et al.*, 2012; Neupane *et al.*, 2018; however see Buyuklu *et al.*, 2009 for contradictory findings). While our results showed changes in VEMPs asymmetry following exposure to vection in VR, we did not find a correlation between VEMPs amplitude and motion sickness susceptibility in our sample. Caution is required in interpreting null results and we note that motion sickness susceptibility in our sample was low (8 participants) or moderate (12 participants). Thus, we cannot exclude that this might explain the absence of correlation between physiological measures and motion sickness susceptibility. Moreover, future research could consider

whether changes in VEMPs correspond to alterations in levels of Cybersickness induced by vection in VR.

In the present study we found an increase in VEMPs asymmetry ratios following one minute of exposure to vection in VR. Interestingly, asymmetries in vestibular reflexes have been reported in other visuo-vestibular discrepant contexts. For example, changes in VEMPs asymmetry have been described after exposure to altered gravity environments (Clarke & Schönfeld, 2015). In microgravity, the absence of gravitational cues alters vestibular functioning, which may be similar to the absence of vestibular cues during vection in VR. Accordingly, Clarke and Schönfeld (2015) found greater VEMPs asymmetry immediately after individuals returned from a short-term Shuttle mission, with symmetry returning to baseline levels 5-8 days post-flight.

The changes in VEMPs asymmetry ratio in the present study corresponded to a substantial increase in VEMPs amplitude recorded on the left sternocleidomastoid muscle following exposure to VR vection. It is possible that this asymmetry may be related to asymmetries in cortical vestibular, VEMPs, and vection processing. Firstly, the vestibular cortical network is distributed asymmetrically, with a preponderance of vestibular cortical regions on the right hemisphere in right-handed individuals (Dieterich *et al.,* 2003). Thus, differences in vestibular cortical processing might have caused an interaction between vestibular responses and vection conditions. Secondly, VEMPs have been demonstrated to elicit differences in hemispherical cortical activity (Schlindwein *et al.*, 2008). Specifically, both left and right VEMPs activated ipsilateral superior, transverse and middle temporal gyri and posterior insula, however left VEMPs also included a deactivation of bilateral dorsomedial frontal cortex, right postcentral and supramarginal gyrus, and left caudate body and cerebellar tonsil. In addition, right VEMPs activations were comparatively stronger than left VEMPs

activations, potentially reflecting the right hemisphere preponderance previously reported (Dieterich *et al.*, 2003; Schlindwein *et al.*, 2008). Thus, we cannot exclude that these asymmetries in cortical VEMPs processing are further enhanced following exposure to vection in VR. Finally, as well as asymmetries in cortical vestibular processing, asymmetric hemispheric effects have been found in relation to vection processing. Kovács *et al.* (2008) for example found greater activation in right MT+ during self vs object motion perception, as well as greater left precuneus activation. Moreover, several changes relating to visually-induced motion sickness (VIMS) have been found, including a decreased correlation between left and right MT+ activity (Miyazaki *et al.*, 2015), reduced connectivity between left and right V1, and increased connectivity between right MT+ and anterior insula and left MT+ and MCC (Toschi *et al.*, 2017). Taken together, differences in cortical activity induced by VEMPs, vestibular functioning, and vection may account for the differential effects of vection on left versus right VEMPs in the present study, however further verification is necessary.

An extensive account of after-effects of VR exposure has not yet been conducted (Gallagher & Ferrè, 2018). Previous research has found that 20 minutes of exposure to VR has detrimental effects on proprioceptive coordination between eyes, hands, and head (Harm *et al.*, 2008), increased pointing errors (Stanney *et al.*, 1999), and decreases in vestibular-ocular reflex gain (Di Girolamo et al., 2001). Here we found increases in VEMPs asymmetry and amplitude following just one minute of exposure to vection in VR, suggesting that the effects of VR adaptation may occur within the first moments of VR exposure. As participants in the present study were exposed to VR self-motion over a very brief timescale (less than 2 minutes), further changes in VEMPs asymmetry may become apparent after longer exposures to VR as

participants habituate to the sensory conflict. Moreover, while the majority of participants in the present study reported that they felt the sensation of vection, we did not include additional measures of vection qualities, such as its intensity. Future research may therefore consider whether such qualities correlate with modulation of the VEMPs. Furthermore, while we investigated vection in VR, it is possible that similar changes may arise from vection induced by other sources, such as projections or computer screens (Keshavarz *et al.*, 2017). Further research could therefore consider any potential differences in VEMPs according to display type.

VR is predicted to be pervasive in our lives: in five years we will use VR as we are now using smartphones. Although VR is revolutionizing our approach to technologies, education and entertainment, there is a widely recognised need to identify whether such technology can affect neural processing and behaviours (Gallagher & Ferrè, 2018). Our results indicate that vestibular processing is rapidly altered during vection in VR. Importantly, this occurs below the user's conscious perception and might explain the after-effects often reported after VR exposure (Stanney *et al.*, 1999; Di Girolamo et al., 2001).

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Chapter 6:

Can Cybersickness in Virtual Reality be Reduced by Decreasing

Vestibular Reliability?

Gallagher, M. & Ferrè, E. R. (In Preparation). Can Cybersickness in Virtual Reality be Reduced by Decreasing Vestibular Reliability?

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Abstract

Virtual Reality (VR) has gained in popularity in recent years. However, despite technological improvements, a significant proportion of VR users may experience motion sickness symptoms, called *cybersickness*. Although the causes of cybersickness are still debated, it is likely to be triggered by visuo-vestibular conflicts for self-motion perception. Under normal circumstances, visual and vestibular cues are integrated according to their reliability, with more reliable senses given a higher weighting. In VR, visual cues signal that the user is moving while vestibular cues signal that the user is stationary. In order to adapt to the VR environment, vestibular cues may be down-weighted, reducing the sensory conflict. Thus, inducing vestibular down-weighting may be a potential method of cybersickness prevention. We have hypothesised that reducing the reliability of vestibular afferent signals might reduce cybersickness symptoms in VR. To achieve a reduction of vestibular reliability, we passively placed participants in a pitch-tilted body orientation. Critically, this manipulation can be implemented in a wide range of VR users, and requires no modification of the normal VR setup. While participants experienced cybersickness symptoms, no difference was found between different body orientations.

Keywords: Virtual Reality, Cybersickness, Multisensory Integration, Vestibular System

Introduction

Virtual Reality (VR) is proving a compelling technology in a range of sectors, from gaming and entertainment, to rehabilitation and training. In recent years, substantial technological advancements in VR have been achieved: display resolution and frame rates have nearly doubled from the first development head-mounted displays (HMDs) to the present commercially available devices (Shafer, Carbonara, & Korpi, 2017, 2019). However, despite clear advances in display technology, significant barriers to VR use and enjoyment remain. One key problem with VR is the occurrence of cybersickness; symptoms of nausea, disorientation, and oculomotor problems which may occur in up to 80% of users (Barrett, 2004; Rebenitsch & Owen, 2016; Stanney, Kennedy, & Drexler, 1997). Although the causes of cybersickness are still debated (Bles, Bos, De Graaf, Groen, & Wertheim, 1998; Dennison & D'Zmura, 2017; Rebenitsch & Owen, 2016; Riccio & Stoffregen, 1991), it seems plausible that it is triggered by conflicts between sensory signals providing self-motion information (Bos, Bles, & Groen, 2008; Reason & Brand, 1975). When moving through the real world, signals from both vision and the vestibular system provide corroborating information regarding speed and direction. Accordingly, these senses are integrated to provide a coherent percept of self-motion (Angelaki, 2014; DeAngelis & Angelaki, 2012). However, in VR, visual and vestibular signals may provide contradictory information, which may affect the way in which sensory signals are usually integrated. Consider a typical VR scenario, such as a rollercoaster. While immersed in the VR rollercoaster, vision provides compelling information that you are moving through space in a certain direction with a certain acceleration. However vestibular signals do not corroborate this visual representation, given that you may actually be stationary. Thus, this visuo-vestibular conflict may lead to symptoms of cybersickness (Bos et al.,

2008; Keshavarz & Hecht, 2011a; Keshavarz, Riecke, Hettinger, & Campos, 2015; Reason & Brand, 1975). Accordingly, in order to prevent cybersickness from occurring, the brain must habituate to ignore the irrelevant vestibular cues, and instead extract self-motion cues from vision alone (Gallagher & Ferrè, 2018; Weech & Troje, 2017). This dynamic vestibular reweighting could therefore be a target for cybersickness prevention.

Bayesian frameworks for multisensory integration stipulate that senses are weighted according to their reliability, and successful sensory integration reduces uncertainty and noise regarding the source percept (Ernst & Banks, 2002; Knill & Pouget, 2004). Thus, greater weight is placed on more reliable senses, and bimodal estimates are more precise than unimodal estimates (Ernst & Banks, 2002; Knill & Pouget, 2004). Visuo-vestibular integration appears to follow the predictions of Bayesian optimal integration, with greater precision when both visual and vestibular cues are available for heading direction, and increased weight placed on vestibular cues when vision becomes unreliable (Angelaki, Gu, & DeAngelis, 2011; DeAngelis & Angelaki, 2012; Fetsch, Turner, DeAngelis, & Angelaki, 2009). During VR, visual cues signal that the user is moving, while vestibular cues signal that the user is stationary. This sensory conflict may be an underlying cause of cybersickness symptoms, with higher levels of sickness reported when greater discrepancies between visual and vestibular cues are apparent (Bonato, Bubka, & Palmisano, 2009; Keshavarz & Hecht, 2011a; Lo & So, 2001). In order to adapt to the VR environment and avoid cybersickness, visual and vestibular cues for self-motion may need to be re-weighted. Specifically, vestibular cues may be down-weighted such that self-motion information comes predominantly from vision and the visuo-vestibular conflict

becomes less salient (Gallagher & Ferrè, 2018). This dynamic vestibular reweighting may therefore result in reduced cybersickness.

At present, it is not possible to wholly prevent cybersickness, although several methods have been proposed. In particular, multiple exposures to VR (Hill & Howarth, 2000; Howarth & Hodder, 2008; Regan, 1995), 'rest frames' providing a stable visual reference for the horizon and vertical in the VR scenario (Chang et al., 2013; Han et al., 2011), and physical locomotion through the virtual world (Llorach, Evans, & Blat, 2014) have all shown promise in reducing symptoms of cybersickness. However, these solutions have not been widely adopted, and may entail significant practical problems such as software modification, limits on space, and a reluctance of VR users to undergo multiple exposures following initial sickness. An alternative solution to cybersickness is the prevention of visuo-vestibular conflict. Application of artificial vestibular stimulation during VR simulators has been shown to reduce levels of cybersickness in several studies (Cevette et al., 2012; Reed-Jones, Reed-Jones, Trick, & Vallis, 2007). Specifically, artificial vestibular stimulation, namely Galvanic Vestibular Stimulation, is used to replace absent vestibular cues during VR navigation. Thus, both vision and the vestibular system signal that the user is moving, and visuo-vestibular conflict is minimised (Cevette et al., 2012, Reed-Jones et al., 2007). While this is a promising technique, significant problems remain. Specifically, the correspondence between the perceived virtual rotation elicited by Galvanic Vestibular Stimulation and natural motion is not yet clear, i.e. the precise self-motion parameters of this virtual sensation have not yet been fully identified. Thus, Galvanic Vestibular Stimulation might not be wholly effective in preventing cybersickness as the vestibular cue cannot be precisely matched to visual self-motion signals in VR. In addition, vestibular stimulation itself may entail side-effects such as uncomfortable cutaneous sensations on the skin surface,

or even nausea (Utz et al., 2011), therefore preventing universal application to all VR users. Finally, access to specialist artificial vestibular stimulation devices may not be feasible for all VR users, further limiting its application.

While previous studies have investigated prevention of visuo-vestibular conflict through substitution of vestibular signals (Cevette et al., 2012; Reed-Jones et al., 2007), an alternative method may be the introduction of noise into the vestibular channel. According to optimal integration frameworks, increased noise within a sensory modality reduces its reliability, resulting in down-weighting of the signals in favour of more reliable cues (Ernst & Banks, 2002; Fetsch et al., 2009). As such, introducing noise into the vestibular channel may reduce its reliability, inducing vestibular down-weighting and decreasing visuo-vestibular conflict in VR (Gallagher & Ferrè, 2018). Cybersickness symptoms may therefore be reduced, as the visuo-vestibular conflict is minimised.

Preliminary research has investigated Bone Conducted Vibration on the mastoid processes in order to decrease the reliability of vestibular signals during VR (Weech, Moon, & Troje, 2018). Bone Conducted Vibration transmits vibrations through the mastoids, resulting in small linear accelerations of the otolith organs within the vestibular labyrinth (Curthoys et al., 2014; Rosengren, McAngus Todd, & Colebatch, 2005). Accordingly, vestibulo-ocular and myogenic reflexes are elicited by Bone Conducted Vibration, comparable to those elicited by other established methods of vestibular stimulation such as sound-evoked or electrical stimulation (Curthoys et al., 2014; Rosengren et al., 2005). Interestingly, Bone Conducted Vibration has been shown to reduce the latency of vection, and reduce symptoms of cybersickness in VR (Weech, Moon, & Troje, 2018; Weech & Troje, 2017). While this form of vestibular stimulation may be better tolerated than artificial vestibular stimulation (Curthoys,

Vulovic, & Manzari, 2012), further exploration of its efficacy across different VR scenarios is necessary. Moreover, like artificial vestibular stimulation, Bone Conducted Vibration also requires access to specialist equipment which may not be available to all VR users.

A convenient physiological way to manipulate the reliability of vestibular cues may be changing body orientation with respect to gravitational acceleration, i.e. tilting individuals away from the gravitational vertical (Alberts, de Brouwer, Selen, & Medendorp, 2016; Burns & Blohm, 2010; Ward, Bockisch, Caramia, Bertolini, & Tarnutzer, 2017). In this body orientation, the otolith organs within the vestibular system are no longer aligned with gravity, and therefore may provide noisier estimates of head position with respect to the gravitational vector (Tarnutzer, Bockisch, Straumann, & Olasagasti, 2009; Vimal, DiZio, & Lackner, 2017). Subsequently, the relative reliability of the vestibular signal is reduced in favour of other sensory information regarding the gravitational vertical, which may result in down-weighting of vestibular cues in favour of visual cues. Accordingly, Alberts et al. (2016) demonstrated that when the head was roll-tilted 30° participants relied more on visual cues for estimating the subjective visual vertical, suggesting a down-weighting of gravitational cues from the vestibular organs. Given these findings, it may therefore be possible to down-weight vestibular cues and reduce cybersickness by exposing participants to VR while they are slightly tilted away from the gravitational vertical.

Here we investigated whether tilting participants during VR exposure would lead to lower cybersickness scores. Participants viewed a VR rollercoaster while upright or tilted. We measured participants' level of cybersickness both during and after the VR scenario, as well as physiological responses to cybersickness, such as heart rate (Kim, Kim, Ko, & Kim, 2001; Nalivaiko, Davis, Blackmore, Vakulin, &

Nesbitt, 2015). We predicted lower cybersickness and heart rate for participants who were tilted versus those who were upright. Critically, this manipulation is simple and cost-effective, as it does not require access to specialist equipment or modifications of VR software or hardware, unlike other techniques previously studied. Accordingly, if this manipulation can decrease symptoms of cybersickness, it may have significant positive impacts on the VR user experience with limited negative side-effects.

Methods

Ethics

The experimental protocol was approved by the Royal Holloway University of London ethics committee. Written informed consent was obtained from all participants prior to completing the study. The study was conducted in line with the Declaration of Helsinki.

Participants

Twenty-four participants (19 female, mean age = 23.08, SD = 2.67) completed the study. Twenty-three participants were right-handed according to their Edinburgh Handedness Inventory questionnaires (Oldfield, 1971). Exclusion criteria were any history of neurological, psychiatric, or vestibular conditions, epilepsy or family history of epilepsy. All participants' data was included in the analysis, resulting in a total sample size of 24 participants.

Experimental Procedure

After completing informed consent procedures, participants were given task instructions. Participants were then asked to step onto a 3D inversion table and wear a

heart rate monitor worn on the wrist (Mio ALPHA 2 smart watch, Mio Technology, Taipei, Taiwan). Half of participants were assigned to the Upright condition, with the remaining half assigned to the Tilted condition. Participants in the Upright condition maintained an upright posture, such that the vestibular organs were congruent with the direction of gravity. Participants in the Tilted condition leant back on the inversion table such that the body axis was tilted 40° backwards from the gravitational vector (Figure 1A).

Once participants were in the correct posture, the VR scenario was started. A custom rollercoaster built using NoLimits 2 software (Lange, 2018) was played on an Oculus Rift CV1 Head-Mounted Display (HMD). The rollercoaster featured accelerations and decelerations, as well as rotations in roll, pitch, and yaw. The speed of the rollercoaster varied between approximately 5.5-50 m/s according to the rollercoaster track. Participants passively viewed the rollercoaster scenario from a first-person perspective. The scenario lasted approximately 10 minutes, unless the participant requested that it was stopped. None of the participants had previously seen the scenario.

To capture participants' level of cybersickness throughout the duration of the scenario, the Fast Motion Sickness scale (FMS) was used (Keshavarz & Hecht, 2011b). This scale requires participants to give a rating of nausea on a scale from 0 to 20, where 0 is no nausea at all and 20 is frank sickness. Participants were asked to give these ratings once before commencing the VR scenario, every 60 seconds during the scenario, and once immediately following the scenario.

Participants' heart rate was monitored throughout the VR scenario. Heart rate has been shown to increase with greater levels of sickness in VR (Kim et al., 2001; Nalivaiko et al., 2015). Participants wore a smart watch which provided continuous

readings of heart rate, with measurements recorded at the same time as the FMS ratings. Thus, heart rate was recorded once prior to commencing the VR scenario, every 60 seconds during the scenario, and once immediately following the scenario.

Following completion of the VR scenario, participants completed the Simulator Sickness Questionnaire (SSQ) following the end of the scenario (Kennedy, Lane, Berbaum, & Lilienthal, 1993). The SSQ captures a total sickness score (SSQ-T), as well as subscale scores of oculomotor (SSQ-O), nausea (SSQ-N), and disorientation (SSQ-D) symptoms.

Data Analysis

Independent t-tests were used to assess whether baseline values of heart rate and FMS differed between Upright and Tilted groups. No significant differences were found ($p > .05$), therefore analyses were conducted on the raw FMS and heart rate values. A 2 x 3 mixed ANOVA with Body Orientation (Upright vs Tilted) and Time (Pre-VR, Peak During VR and Post-VR) as factors were used to analyse FMS and heart rate. Bonferroni-corrected post-hoc tests were used to examine significant main effects and interactions.

In addition, for each participant, the slope of the linear regression trendline was estimated for FMS and heart rates across the 10 during-VR timepoints. Independent t-tests with Body Orientation as the grouping variable were then applied to these values. Similar independent t-tests were also conducted on SSQ total (SSQ-T), nausea (SSQ-N), oculomotor (SSQ-O) and disorientation (SSQ-D) subscales.
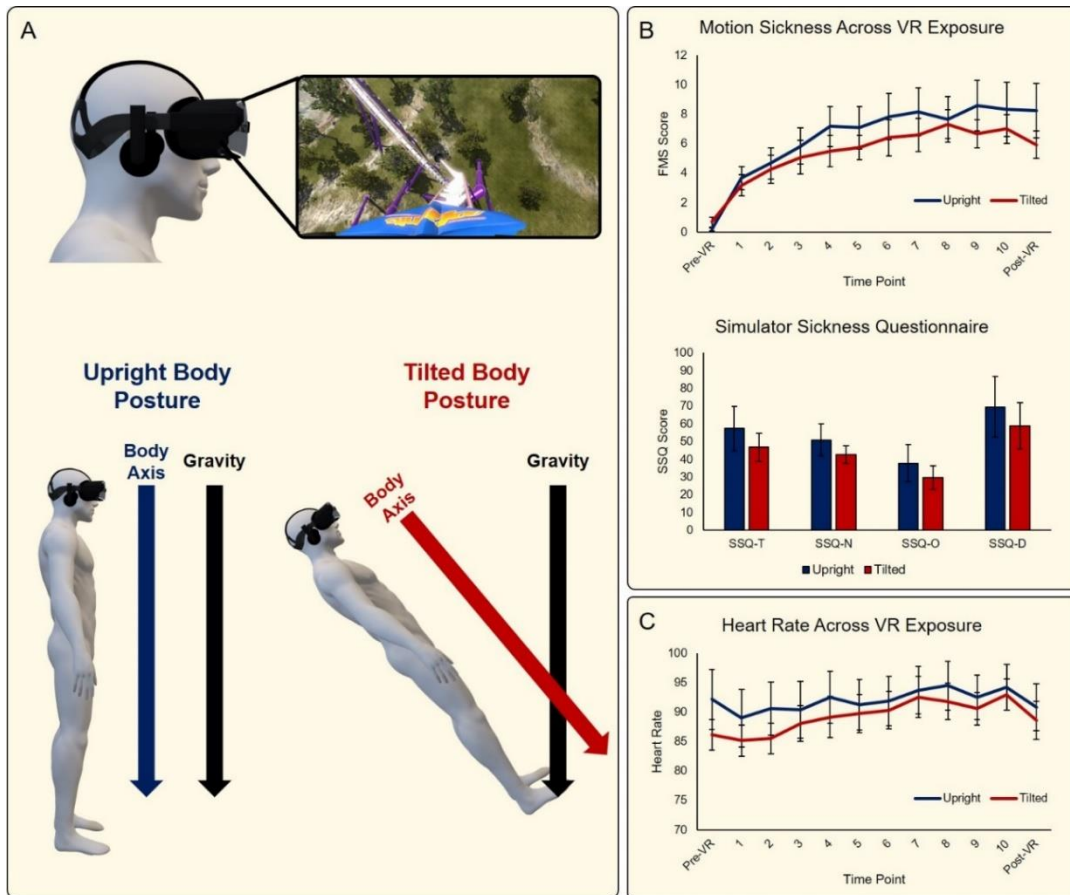
Figure 1. Experimental set up and results. A) VR scenario and body postures. B) FMS across time (top) and SSQ (bottom) results. C) Heart rate across time.

Results

Fast Motion Sickness Ratings

FMS scores across VR exposure can be seen in Figure 1B. The 2x3 ANOVA revealed a significant main effect of Time ($F(1.40, 30.86) = 51.99$, $p < .001$, $\eta_p^2 = .70$). Post-hoc tests showed that FMS scores were significantly higher during VR (mean = 9.13, SE = 0.96) relative to pre-VR (mean = 0.42, SE = 0.18) ($p < .001$) and post-VR (mean = 7.08, SE = 1.04) ($p = .003$). In addition, post-VR FMS scores were significantly higher than pre-VR scores ($p < .001$). Thus, FMS scores increased during exposure to the VR rollercoaster and decreased slightly following exposure to VR, but

remained higher than pre-VR values. Individual data points for pre- peak- and post-VR FMS scores can be seen in Appendix 4, Figure A10.

No significant main effect of Body Orientation was found ($F(1, 22) = 0.34$, $p = .57$, $\eta_p^2 = .02$). No interaction between Time and Body Orientation was found ($F(1.40, 30.86) = 1.31$, $p = .28$, $\eta_p^2 = .06$).

The independent t-test revealed no significant difference in FMS slopes during VR between Body Orientations ($t(22) = 0.87$, $p = .40$). Individual data points for FMS slopes are plotted in Appendix 4, Figure A11. All raw FMS data are available in Appendix 5, Table A7.

Simulator Sickness Questionnaire

SSQ results can be seen in Figure 1B. The independent t-tests revealed no significant effect of Body Orientation on SSQ-T ($t(22) = 0.71$, $p = .48$), SSQ-N ($t(22) = 0.79$, $p = .44$), SSQ-O ($t(22) = 0.66$, $p = .52$), or SSQ-D scores ($t(22) = 0.59$. $p = .56$). Individual data points are plotted in Appendix 4, Figure A12, while raw data are available in Appendix 5, Table A6.

Heart Rate

Heart rate across time can be seen in Figure 1C. The 2x3 ANOVA revealed a significant main effect of Time on heart rate ($F(2, 44) = 23.27$, $p < .001$, $\eta_p^2 = .51$). Post-hoc tests showed that peak heart rate was significantly higher during VR (mean = 97.13, SE = 2.73) relative to pre-VR (mean = 89.17, SE = 2.86 ($p < .001$) and post-VR (mean = 89.71, SE = 2.56) ($p < .001$). No significant difference in pre- and post-VR heart rate was found ($p > .99$). Thus, heart rate increased during exposure to the VR rollercoaster and returned to pre-VR values immediately following exposure.

No significant main effect of Body Orientation was found ($F(1, 22) = 0.49$, $p = .49$, $\eta_p^2 = .02$). No significant interaction was found between Time and Body Orientation ($F(1, 44) = 1.22$, $p = .31$, $\eta_p^2 = .05$). Individual data points for pre- peak- and post-VR heart rate can be seen in Appendix 4, Figure A13.

The independent t test revealed a significant difference in heart rate slopes between upright and tilted Body Orientations ($t(16.32) = -2.69$, $p = .02$, Cohen's $d = 1.10$). The mean slope for the upright orientation was 0.08 (SD = 0.54), versus 0.55 (SD = 0.28) for the tilted orientation. This difference may be driven by lower heart rates for the tilted participants during the first three during-VR time points, although individually these timepoints did not differ significantly ($p > .05$). Individual data points for heart rate slopes are plotted in Appendix 4, Figure A14, while all raw heart rate data are available in Appendix 5, Table A8.

Discussion

Cybersickness remains a significant barrier to VR use, despite advances in VR technology (Shafer et al., 2017, 2019). Proposed techniques to prevent symptoms from developing include rest frames, repeated exposure, locomotion, and artificial vestibular stimulation (Cevette et al., 2012; Chang et al., 2013; Hill & Howarth, 2000; Llorach et al., 2014; Reed-Jones et al., 2007). However, no single technique has had widespread adoption. While technological solutions such as artificial vestibular stimulation or Bone Conducted Vibration appear promising, they come with intrinsic limitations. For instance, adverse side effects in some users and a lack of widespread availability. Thus, non-technological solutions to the problem of cybersickness may be necessary.

The underlying cause of cybersickness is likely to be visuo-vestibular conflict (Bles et al., 1998; Bonato et al., 2009; Reason & Brand, 1975; Rebenitsch & Owen, 2016). To habituate to the VR environment, vestibular cues may be down-weighted such that self-motion information comes predominantly from vision (Gallagher & Ferrè, 2018). Consequently, visuo-vestibular conflict is minimised, and lower levels of cybersickness may be experienced. Accordingly, previous research suggests that reducing the reliability of the vestibular system by introducing noise can result in lower levels of cybersickness (Weech et al., 2018). However, this method of cybersickness prevention may rely on technology which is unavailable to all VR users, and may not be practical in all contexts. Interestingly, vestibular cues may be physiologically and non-invasively downweighted when individuals are tilted away from the gravitational vertical (Alberts et al., 2016; Ward et al., 2017). Here, we investigated whether tilting participants during exposure to a VR rollercoaster would reduce levels of cybersickness. As expected, we found increased levels of cybersickness across time, however no significant differences were found between upright and tilted postures.

Previous research suggests that levels of cybersickness tend to increase with longer durations of exposure to VR (D'Amour, Bos, & Keshavarz, 2017; Keshavarz & Hecht, 2011b; Liu, 2014). Accordingly, FMS ratings show continuous increases across each minute of VR exposure (D'Amour et al., 2017), while SSQ total scores are higher at 15 minutes versus 5 minutes of VR (Liu, 2014). Importantly, physiological measures of cybersickness also show similar increases over time. Dennison, Wisti, & D'Zmura (2016) reported that both subjective ratings of cybersickness and average heartbeats per minute increased dramatically from pre-VR levels throughout 10 minutes of VR exposure, with a slight decrease after exiting VR. Accordingly, we found increases in FMS scores and average heart rate across 10 minutes of VR

exposure, further highlighting the effect of exposure duration on levels of cybersickness.

While we found a significant effect of time, body orientation appeared to have little impact on levels of cybersickness. Why did our modulation not successfully reduce cybersickness? Although caution is needed when commenting on null results, we have speculated on different options. The vestibular system signals complex and dynamic movement in 3D space (Britton & Arshad, 2019; Dichgans & Brandt, 1978; Soyka, Bülthoff, & Barnett-Cowan, 2015). Specifically, the semicircular canals signal angular rotations in roll, pitch, and yaw, while the otolith organs signal linear acceleration from translation and gravity. When tilted away from the gravitational vertical, otolith cues for posture with respect to gravity may become unreliable, and down-weighted in favour of other cues for gravity (Vimal et al., 2017). However, dynamic semicircular canal cues for self-motion may still be reliable. Thus, we cannot rule out that these semicircular canal afferents were still implicated in processing self-motion during exposure to VR, even if otolith cues were down-weighted when the body was tilted. Importantly, our VR scenario included both linear translation and rotation in roll, pitch, and yaw. Accordingly, it may be the case that conflicts between semicircular canal afferents and visual rotations may have increased levels of cybersickness, accounting for the lack of change according to posture.

In our previous work, we found that vestibular processing was altered after exposure to short duration vection (Gallagher, Choi, & Ferrè, Under Review). Specifically, we found that participants were less able to detect cues from artificial vestibular stimulation after viewing a vection-inducing stimulus in VR. Crucially, this was only the case when both vection and vestibular cues were congruent, suggesting that vestibular down-weighting occurred only for the specific plane of visual motion.

Given that the VR scenario in the present study included both linear acceleration and rotations in roll, pitch, and yaw, it may be possible that the down-weighting of otolith cues through tilting participants was not sufficient to reduce cybersickness in a complex 3D VR scenario. While we predicted that a 40° pitch-tilt would down-weight vestibular cues for self-motion, further studies may be necessary to verify this, and to fine-tune parameters for vestibular down-weighting through posture modification.

Interestingly, we found a significant difference in slopes for heart rate during VR exposure. Specifically, slopes were greater for tilted versus upright participants. Visual inspection of the data suggested that this increase in slope was driven by lower heart rate in tilted participants during initial VR exposure, whereas heart rate was similar across all time points for upright participants. Importantly, the vestibular system shows rapid adaptation in response to constant accelerations and static tilts (Eron, Cohen, Raphan, & Yakushin, 2008; Fernandez, Goldberg, & Abend, 1972; St George, Day, & Fitzpatrick, 2011). Accordingly, the firing rate of vestibular neurons and the vestibulo-ocular reflex decline when exposed to constant centrifugal forces and rotations respectively (Fernandez et al., 1972; St George et al., 2011). Given that we did not modify body orientation throughout VR exposure, it is therefore possible that participants habituated to the tilted posture over time. Symptoms of cybersickness are likely to develop with increasing exposure to VR (Liu, 2014; Moss et al., 2011; Stanney, Kingdon, Graeber, & Kennedy, 2002). Accordingly, in the present study we found significant effects of time on FMS scores, with greater levels of cybersickness during and after the VR scenario. As such, it is possible that adaptation to the tilted posture in the first minutes of exposure may have masked any potential changes in cybersickness scores. Thus, it may be possible that only continuous noise added to the

vestibular cue, such as Bone Conducted Vibration (Weech et al., 2018), can reduce vestibular reliability sufficiently across the entire duration of VR exposure.

Several limitations to our study must be taken into account. Firstly, we used a between-subjects design in order to avoid carry-over effects from repeated exposure. Importantly, a pilot study suggested significant carry-over effects when a within-subjects design was used. While we took care to ensure that baseline values of FMS and heart rate did not differ between the two groups, we cannot rule out that the participants within each group responded differently to the VR scenario. Moreover, a within-subjects design would have allowed a greater sample size for each experimental condition. In principle, the null result may reflect low statistical power. We performed a power calculation to estimate the number of participants required to obtain a significant difference between body orientations. The sample size calculation for a 2x3 mixed ANOVA was based on previously reported effect sizes (Weech et al., 2018), with alpha = .05 and power = 0.95, giving a total sample of 18 participants. Thus, while we believe our study was adequately powered, future research should consider whether a within-subjects design could potentially reveal significant differences between upright and tilted body orientations.

Secondly, we used the FMS to assess changes in nausea symptoms during VR exposure. While this scale was designed to capture fine-grained changes in sickness across time, it cannot account for changes in other symptoms such as disorientation or fatigue (Keshavarz & Hecht, 2011b). Although we used the SSQ (Kennedy et al., 1993) to assess these additional symptoms, this was only administered following conclusion of the VR scenario. Accordingly, it is possible that more subtle changes in these non-nausea symptoms over time may not have been captured by our cybersickness measures. Previous research varied the duration of VR exposure in

different blocks in order to assess SSQ scores (Liu, 2014). Thus, future research may consider this approach to more precisely assess alternative symptoms of cybersickness during VR.

Finally, as discussed previously, it may be possible that participants adapted to the posture over time, masking changes in cybersickness throughout VR exposure. We decided on an arbitrary static posture based on previous findings reporting re-weighting of vestibular cues with body tilt (Alberts et al., 2016; Burns & Blohm, 2010). Future research should therefore explore firstly whether increased angles of tilt or tilt on other axes would be more efficient in reducing vestibular weighting, and secondly whether dynamically modifying posture could reduce levels of cybersickness.

Overall, here we found no significant effect of posture on symptoms of cybersickness. We found a significant difference in heart rate slopes, which suggested that heart rate was lower for tilted participants during the initial exposure to VR before increasing with time. This suggested therefore that a degree of habituation to the tilted posture had occurred, which could potentially mask reductions of cybersickness as these symptoms typically develop with increasing exposure. While previous research suggests a down-weighting of vestibular cues when tilting the head relative to gravity, it is possible that semicircular canal cues for self-motion were not down-weighted, accounting for the lack of change in cybersickness scores in the tilted participants. Future research could consider within-subjects designs, alternative measures of cybersickness, alternative static tilts, or dynamic modification of posture to address the limitations of our study.

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Chapter 7:

Vestibular Stimulation in Virtual Reality: Is Galvanic Vestibular Stimulation a method to reduce Cybersickness?

Gallagher, M., Thorn, J., Slater, M., & Ferrè, E. R. (In Preparation). Vestibular Stimulation in Virtual Reality: Is Galvanic Vestibular Stimulation a method to reduce Cybersickness?

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Abstract

Virtual Reality (VR) is gaining in popularity across a wide range of sectors. However, a significant barrier to VR uptake is the occurrence of motion sickness symptoms, known as *cybersickness*. It is likely that conflicts between visual and vestibular cues for self-motion are the underlying cause of symptoms. Specifically, while vision signals that the user is moving through the VR environment, vestibular cues signal that the user is stationary. Thus, cybersickness may be reduced by artificial vestibular stimulation in order to replace vestibular cues for self-motion. In the present study we used Galvanic Vestibular Stimulation (GVS) to reduce sensory conflict during a VR driving simulator. Participants completed a driving scenario with GVS or Sham stimulation. We found no change in cybersickness following exposure to VR with or without GVS. Further research may be necessary to fine-tune GVS for prevention of VR sensory conflicts.

Keywords: Virtual Reality, Galvanic Vestibular Stimulation, Cybersickness, Sensory Conflict

Introduction

Virtual Reality (VR) is proving a great benefit in a range of sectors, from gaming and recreation to rehabilitation and training. In recent years, technological advancements have resulted in VR becoming widely available via a range of commercial headsets (Shafer, Carbonara, & Korpi, 2017, 2019). However, despite advances in technology, a significant proportion of VR users will experience motion sickness symptoms such as nausea, disorientation, and oculomotor disturbances (Barrett, 2004; Rebenitsch & Owen, 2016; Stanney, Kennedy, & Drexler, 1997). *Cybersickness* remains a barrier to the full potential of VR uptake. Critically, at present there is no way to predict which users are likely to experience symptoms, nor are there reliable methods of prevention (LaViola, 2000; Rebenitsch & Owen, 2014).

Many VR applications utilise visual information to give the user a compelling sense of motion through the virtual world. Consider a typical VR scenario in which the user is driving a car: buildings and trees moving past the retina signal that the user is moving in a certain direction with a certain acceleration. However, use of optic flow, while inducing the illusion of vection, also results in a conflict between visual and vestibular cues for self-motion – although vision signals that the user is moving, the vestibular cues signal that the user is actually stationary. This visuo-vestibular conflict is likely to be the underlying cause of cybersickness (Bles, Bos, De Graaf, Groen, & Wertheim, 1998; Keshavarz, Riecke, Hettinger, & Campos, 2015; Oman, 1988; Reason & Brand, 1975). Accordingly, Bonato, Bubka, and Palmisano (2009) and Keshavarz and Hecht (2011) described high levels of cybersickness when users were exposed to optic flow while in a stationary posture. Interestingly, increasing the sensory conflict between visual and vestibular cues by adding a second axis of visual

motion exacerbated cybersickness symptoms (Bonato et al., 2009; Keshavarz & Hecht, 2011).

In order to reduce cybersickness, one might need to minimise visuo-vestibular conflicts in VR. One way in which this may be achieved is by allowing VR users to physically navigate through the real world while viewing a VR scenario (Llorach, Evans, & Blat, 2014; Whitton et al., 2005). In such cases, visuo-vestibular conflict is minimised as visual information from the virtual environment is matched with vestibular information from physical motion. Accordingly, Llorach, Evans and Blat (2014) found that cybersickness was significantly lower when individuals navigated the VR environment by walking through physical space with their movements tracked via position trackers, compared to navigation via game controllers. However, locomotion through a virtual environment is not necessarily a practical solution to cybersickness: it is likely to require large spaces or software modifications which may not be ideal for the majority of VR contexts (Williams et al., 2007).

A possible alternative to physical locomotion in VR is artificially generating vestibular self-motion sensations that match with the vection cues provided by the VR environment. Critically, artificial vestibular stimulation may replace the absent vestibular cues in VR scenarios which induce vection through optic flow without the need for large spaces for physical navigation. A widely used method of artificial vestibular stimulation is Galvanic Vestibular Stimulation (GVS) in which electrodes attached to the mastoids deliver a low-intensity electrical current to stimulate the vestibular nerve (Curthoys & MacDougall, 2012; Fitzpatrick & Day, 2004; Stephan et al., 2005). This stimulation results in an illusory sensation of roll rotation (Cathers, Day, & Fitzpatrick, 2005; Day & Fitzpatrick, 2005; Fitzpatrick, Marsden, Lord, & Day, 2002). Thus, the illusory sensations of motion could be used to replace absent

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vestibular sensations of self-motion during VR (Cevette et al., 2012; Maeda, Ando, & Sugimoto, 2005).

Previous research into simulator sickness, a related form of motion sickness present following exposure to non-VR simulators, has reported promising effects of GVS (Cevette et al., 2012; Reed-Jones, Reed-Jones, Trick, & Vallis, 2007). Reed-Jones et al. (2007) investigated whether GVS could reduce simulator sickness when participants actively navigated a fixed-base driving simulator. Participants drove for 15 minutes in the simulator with and without GVS. The stimulation was set to each participants' threshold (between 0.6-1.25 mA of intensity) and was applied during sharp and gradual turns in the virtual environment. Overall simulator sickness levels and symptoms of disorientation were significantly reduced when participants completed the simulator with GVS compared to the same simulator without GVS (Reed-Jones et al., 2007). Similarly, Cevette et al. (2012) investigated whether coupling visual and vestibular rotation cues using GVS could reduce simulator sickness in an active flight simulator. GVS from 1.5 to 2.5 mA of intensity was applied to produce sensations of roll, pitch, and yaw. These rotations were matched to the visual motion presented on the simulator as participants flew the virtual plane. A control group of participants completed the same flight simulator with 1mA of constant GVS, which the authors reported did not elicit comparable motion sensations to the experimental condition. Crucially, symptoms of simulator sickness were significantly reduced in participants who flew the simulator with GVS matching the visual cues versus those who flew with constant GVS. Despite these promising findings, GVS has not yet been applied as a method of reducing cybersickness in an immersive VR scenario.

As well as cybersickness, exposure to VR may result in aftereffects such as poor coordination, increased disorientation, and altered vestibular functioning (Di Girolamo et al., 2001; Harm, Taylor, Reschke, Somers, & Bloomberg, 2008; Stanney & Kennedy, 1998). A comprehensive account of VR-induced aftereffects has not yet been conducted, however it seems likely that these aftereffects arise from adaptation to visuo-vestibular conflicts in VR (Gallagher & Ferrè, 2018). Accordingly, Di Girolamo et al. (2001) found that vestibulo-ocular reflex gain was significantly lower following VR exposure. Similarly, Gallagher, Dowsett, and Ferrè (2019) found an increase in vestibular-evoked myogenic potential asymmetry following 1 minute of exposure to vection in VR. Thus, it is possible that prevention of visuo-vestibular conflicts in VR could not only prevent cybersickness, but also avoid subsequent VR aftereffects.

Here we explored whether application of GVS during VR would reduce levels of cybersickness and possibly prevent VR aftereffects. We measured participants' levels of cybersickness and vestibular reflexes (vestibular-evoked myogenic potentials, VEMPs) after exposure to a VR driving simulator which embedded motion-congruent, short duration, low intensity GVS pulses. Based on preliminary findings (Cevette et al., 2012; Reed-Jones et al., 2007), we expected to replicate lower levels of cybersickness during vestibular integrated VR. Importantly, we also applied sham stimulation to control for non-specific effects of vestibular stimulation.

Methods

Ethics

The experimental protocol was approved by the Royal Holloway University of London ethics committee. The study was conducted in line with the Declaration of

Helsinki. Written informed consent was obtained from the participants before commencing the experiment.

Participants

Fifteen participants (11 female, mean age = 21.33, SD = 3.33) completed the study. All were right-handed according to their Edinburgh Handedness Inventory (Oldfield, 1971) scores. Exclusion criteria were any history of neurological, vestibular, or psychiatric conditions, epilepsy or family history of epilepsy. Data from two participants was excluded due to procedural recording errors in VEMPs data, resulting in a total sample size of 13 participants for analysis.

Sensory Integrated VR-GVS Scenario

A custom sensory integrated VR+GVS scenario rendered in Unity 3D (Unity Technologies 2018) was presented to participants on an Oculus Rift CV1 Head-Mounted Display (HMD). The scenario consisted of a driving simulation in which a car drove participants around a virtual city. Participants were seated at the driver's side of the car and could see clearly out of the front and side windows. The car travelled along a set path and maintained the same speed throughout the 17 left and 17 right turns. Thus, participants passively navigated the virtual environment.

Importantly, this VR scenario embedded a trigger to GVS. GVS was administered by a commercial stimulator (Good Vibrations Engineering Ltd., Nobleton, ON, Canada). Electrodes measuring approximately 4cm^2 were coated with NaCl electrode gel and placed on the mastoids (GVS condition) or on the base of the neck, approximately 5cm below the ear (Sham condition). When the virtual car turned left or right, a 1 second boxcar pulse of 1 mA stimulation was applied. Specifically, a

left-anodal/right-cathodal GVS configuration was applied during leftward turns while a right-anodal/left-cathodal configuration was applied during rightward turns (Figure 1A). GVS induces a sensation of rotation towards the cathodal side, thus the sensation of vection was always congruent with the turns. Sham stimulation was also administered as a control. Stimulation was applied via the electrodes placed on the base of the neck. This stimulation therefore elicits similar cutaneous sensations as GVS without sensations of motion. The total duration was approximately 10 minutes. Participants were instructed to maintain a stable head position throughout the duration of the VR scenario, and to continue focusing directly ahead of them.

Cybersickness Measures

To assess levels of Cybersickness, participants completed the Simulator Sickness Questionnaire (SSQ) (Kennedy, Lane, Berbaum, & Lilienthal, 1993) following conclusion of the VR scenario. This questionnaire divides symptoms of sickness into components of Nausea (SSQ-N), Disorientation (SSQ-D) and Oculomotor (SSQ-O) clusters, as well as a Total (SSQ-T) score. Higher scores therefore corresponded to greater levels of sickness.

VEMPs Recording

To assess changes in vestibular functioning following VR, VEMPs were measured before and after exposure to the VR scenario. VEMPs were recorded according to standard procedures (Colebatch, Halmagyi, & Skuse, 1994; Fowler, Sweet, & Steffel, 2014) using BioMed eVEMP USB software and hardware (BioMed Jena GmbH, 2016). Electrodes were placed on the left and right sternocleidomastoid muscles in a bipolar configuration, with ground electrodes on the forehead and collar

bone. VEMPs were elicited by 500 Hz tone burst stimuli at 100 dB sound pressure level, with a duration of 7 ms. Auditory stimulation was presented via HAD 280 Sennheiser headphones, and was triggered only when the participants' muscle contraction was between 120 and 400 µV RMS and electrode impedance was less than 20 kΩ. Muscle contraction was achieved by asking participants to turn the head to the contralateral side of measurement and to push the head backwards towards the chair while seated comfortably. Participants were asked to maintain this posture and muscle tension through the duration of the measurement. Measurements were recorded at 2000 Hz sampling frequency, and 100 single trials of 80 ms duration were averaged to give the final VEMP measurement. The final VEMP measurements were obtained within approximately 90 seconds.

VEMP amplitudes and latencies were provided automatically by the eVEMP software. Asymmetry ratios were calculated accordingly:

$$Asymmetry\ ratio = \frac{|P1N1Amp_R| - |P1N1Amp_L|}{|P1N1Amp_R| + |P1N1Amp_L|} \times 100$$

Thus, negative asymmetry ratios indicated higher amplitudes on the left side while positive values indicated higher amplitudes on the right side.

Procedure

A within-subjects design was used, with participants experiencing the driving scenario with both GVS and Sham stimulation in separate sessions. The sessions were separated by one week to minimise carry-over effects, and both sessions took place at the same time of day. The order of stimulation type was counterbalanced across participants. The procedure of both sessions was otherwise identical.

After completing informed consent, participants were given task instructions. Both GVS and VEMPs electrodes were then fixed in place, and the participant was asked to sit comfortably on a racing simulator chair. Pre-VR VEMPs measurements were then taken, with the first muscle side of measurement counterbalanced across participants. Following this, the participant wore the HMD and the VR scenario commenced. The scenario lasted for 10 minutes, unless the participant felt too unwell to continue. Immediately following the scenario, the HMD was removed and VEMPs measurements were taken again. Finally, participants completed the SSQ.

Data Analysis

Two participants were excluded from analysis due to procedural errors in recording VEMPs. For VEMPs P1-N1 peak-to-peak amplitudes, P1 and N1 latencies the percentage change between pre- and post-VR measurements was calculated. 2 (Muscle Side: Left vs Right) x 2 (Stimulation: GVS vs Sham) repeated measures ANOVAs were conducted to analyse these data. To analyse VEMPs asymmetry ratios, a 2 (Stimulation: VR+GVS vs VR+Sham) x 2 (Time: Pre vs Post VR) repeated measures ANOVA was used. Paired *t*-tests were also used to analyse differences in total (SSQ-T) and subscale (SSQ-D, SSQ-N and SSQ-O) SSQ scores between VR+GVS vs VR+Sham.
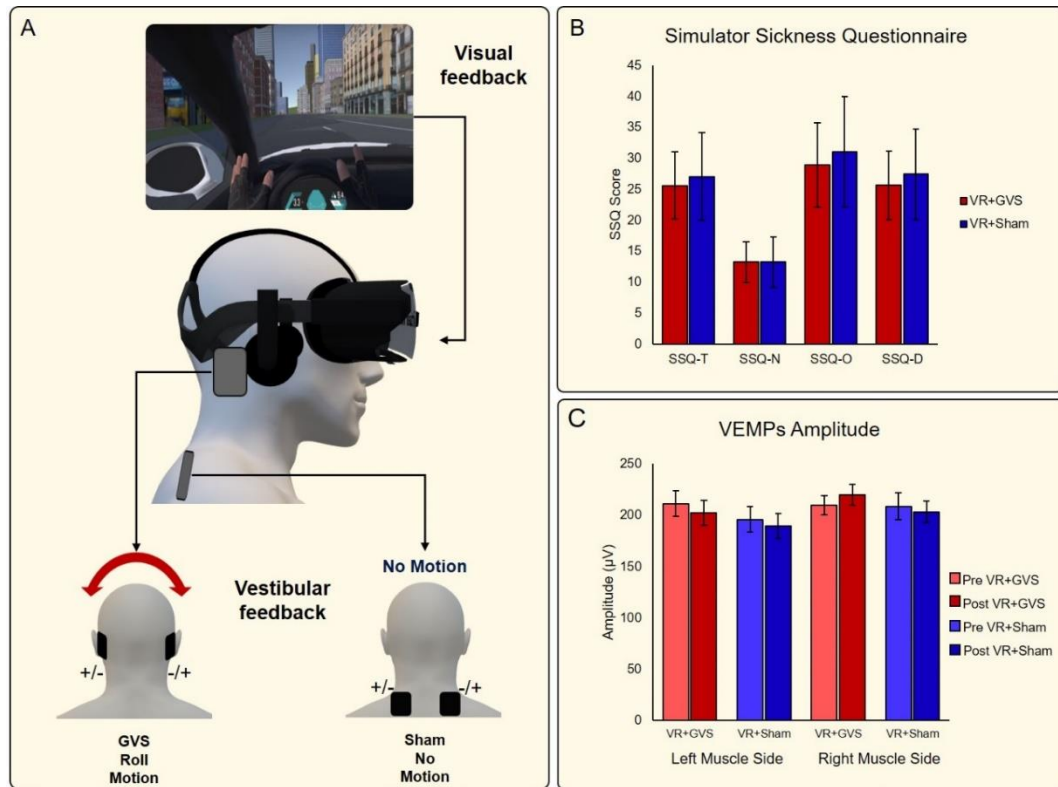
Figure 1. Experiment set up and results. A) Sensory integrated VR-GVS scenario. Participants viewed a VR driving simulator (top) on an Oculus Rift CV1 HMD. Critically, GVS was applied during left and right turns, such that the virtual rotation sensations from GVS matched the visual self-motion in the VR scenario. A sham condition applied stimulation on the base of the neck to control for non-specific effects. B) Simulator Sickness Questionnaire results. C) VEMPs Amplitude results. Error bars represent standard error of the mean.

Results

SSQ Scores

Mean SSQ total and subscale scores can be seen in Figure 1B. No significant difference between VR+GVS and VR+Sham was found on SSQ-T ($t(12)$ = -0.29, p = .78), SSQ-N ($t(12)$ = 0.00, p = 1.00), SSQ-D ($t(12)$ = -0.28, p = .79), or SSQ-O ($t(12)$ = -0.31, p = .76), scores.

To explore whether this null-significance was due to order effects, we conducted a 2 (Stimulation: VR+GVS vs VR+Sham) x 2 (Order: VR+GVS First vs

VR+Sham First) mixed ANOVA, with Stimulation as a within-subjects factor and Order as a between-subjects factor.

For SSQ-T scores, no significant main effect of Stimulation ($F(1, 11) = 0.18$, $p = .68$, $\eta_p^2 = .02$) or Order ($F(1, 11) = 0.57$, $p = .47$, $\eta_p^2 = .05$) was found. No significant interaction between Stimulation and Order was found ($F(1, 11) = 2.39$, $p = .15$, $\eta_p^2 = .18$).

For SSQ-N scores, no significant main effect of Stimulation ($F(1, 11) = 0.03$, $p = .87$, $\eta_p^2 = .003$) or Order ($F(1, 11) = 0.31$, $p = .59$, $\eta_p^2 = .03$) was found. A significant interaction between Stimulation and Order was found ($F(1, 11) = 4.93$, $p = .048$, $\eta_p^2 = .31$), suggesting that participants' SSQ-N scores were lower in their second versus first session across stimulation conditions.

Similar results were found for SSQ-D scores. No significant main effect of Stimulation ($F(1, 11) = 0.25$, $p = .63$, $\eta_p^2 = .02$) or Order ($F(1, 11) = 0.81$, $p = .38$, $\eta_p^2 = .06$) was found. A significant interaction between Stimulation and Order was found ($F(1, 11) = 5.30$, $p = .04$, $\eta_p^2 = .33$), again with lower SSQ-D scores in the second versus first session for both VR+GVS and VR+Sham.

Finally, no significant main effect of Stimulation ($F(1, 11) = 0.10$, $p = .76$, $\eta_p^2 = .009$) or Order ($F(1, 11) = 0.47$, $p = .51$, $\eta_p^2 = .45$) was found on SSQ-O scores. No significant interaction between Stimulation and Order was found ($F(1, 11) = 0.10$, $p = .76$, $\eta_p^2 = .009$). Individual SSQ data points are plotted in Appendix 4, Figure A15, while all raw SSQ data are available in Appendix 5, Table A9.

VEMPs Asymmetry Ratios

Mean VEMPs asymmetry ratios can be seen in Table 1. Individual data points are plotted in Appendix 4, Figure A16, while all raw data are available in Appendix 5, Table A10. The 2x2 ANOVA revealed no significant main effect of Stimulation (F(1, 12) = 0.18, p = .68, η_p^2 = .02), nor of Time (F(1, 12) = 0.55, p = .47, η_p^2 = .04). No significant interaction was found between Stimulation and Time (F(1, 12) = 0.39, p = .54, η_p^2 = .03). Thus, no significant change in VEMPs asymmetry ratios was found following exposure to the VR scenario, irrespective of stimulation type.

Table 1. Mean (SD) VEMPs asymmetry ratios Pre and Post VR+GVS and VR+Sham.

	Pre-VR	Post-VR
VR+GVS	0.11 (9.27)	4.38 (11.80)
VR+Sham	3.06 (12.32)	3.89 (14.14)

VEMPs P1-N1 Peak-to-Peak Amplitude

Mean proportional changes in VEMPs amplitudes across Muscle Sides and Stimulation Types can be seen in Table 2. Mean raw VEMPs amplitudes according to muscle side and VR/GVS condition can be seen in Figure 1C. Individual data points are plotted in Appendix 4, Figure A17, while all raw data are available in Appendix 5, Table A11. No significant main effect of Muscle Side (F(1, 12) = 0.46, p = .51, η_p^2 = .04) or Stimulation (F(1, 12) = 0.09, p = .77, η_p^2 = .008) was found on VEMPs peak-to-peak amplitudes. No interaction between Side and Stimulation was found (F(1, 12) = 0.18, p = .68, η_p^2 = .01). Therefore, no significant differences were observed in VEMPs amplitudes following exposure to VR with or without GVS.

Table 2. Mean (SD) pre-post proportional change in VEMPs amplitudes, P1 and N1 latencies.

	Left Muscle Side		Right Muscle Side	
	VR+Sham	VR+GVS	VR+Sham	VR+GVS
Amplitude	-0.97 (22.45)	-1.59 (23.06)	1.27 (26.19)	5.44 (14.62)
P1 Latency	-0.97 (33.70)	19.27 (60.28)	10.20 (53.38)	-14.43 (20.87)
N1 Latency	4.34 (12.72)	0.88 (12.09)	2.43 (16.12)	1.00 (20.31)

VEMPs P1 and N1 Latencies

Mean proportional changes in VEMPs P1 and N1 latencies across Muscle Sides and Stimulation Types can be seen in Table 2. Individual P1 data points are plotted in Appendix 4, Figure A18, individual N1 data points are plotted in Appendix 4, Figure A19. All raw P1 data are available in Appendix 5, Table A12, while raw N1 data are available in Appendix 5, Table A13. No significant main effect of Muscle Side ($F(1, 12) = 0.55$, $p = .47$, $\eta_p^2 = .04$) or Stimulation ($F(1, 12) = 0.06$, $p = .81$, $\eta_p^2 = .005$) was found on VEMPs P1 latencies. No interaction between Side and Stimulation was found on VEMPs P1 latencies ($F(1, 12) = 2.96$, $p = .11$, $\eta_p^2 = .20$).

Similarly, no significant main effect of Muscle Side ($F(1, 12) = 0.05$, $p = .82$, $\eta_p^2 = .004$) or Stimulation ($F(1, 12) = 0.18$, $p = .68$, $\eta_p^2 = .02$) was found on VEMPs N1 latencies. No interaction between Side and Stimulation was found on VEMPs N1 latencies ($F(1, 12) = 0.08$, $p = .78$, $\eta_p^2 = .007$).

Overall, no significant differences in VEMPs latencies were found following exposure to VR with or without GVS.

Discussion

Cybersickness remains a significant barrier for VR use, however there is currently no way of predicting who will develop symptoms, nor any gold-standard method of prevention (LaViola, 2000; Rebenitsch & Owen, 2014). Many VR applications use optic flow to induce an illusory sense of self-motion, however these visual cues conflict with vestibular cues which indicate that the user is stationary. This visuo-vestibular conflict is likely to be an underlying cause of cybersickness (Barrett, 2004; Gallagher & Ferrè, 2018; Kennedy, Drexler, & Kennedy, 2010). Accordingly, reducing visuo-vestibular conflict may be one method to prevent cybersickness symptoms. Previous research suggests that using artificial vestibular stimulation (i.e., GVS) may reduce simulator sickness by matching visual and vestibular cues for self-motion perception (Cevette et al., 2012; Reed-Jones et al., 2007). However, little research into GVS during VR has been conducted. Moreover, previous research suggests that vestibular processing may be altered following exposure to VR (Di Girolamo et al., 2001; Gallagher et al., 2019). Thus, application of artificial vestibular stimulation may reduce aftereffects of VR exposure. In the present study, we used GVS in a VR driving simulator to investigate whether the stimulation could reduce symptoms of cybersickness. We found no effect of GVS on cybersickness symptoms.

Although previous studies have found reduced simulator sickness when integrating GVS and VR (Cevette et al., 2012; Reed-Jones et al., 2007), several key factors may account for the divergent findings of the present study. Firstly, differences in GVS parameters have been used across the present study and previous simulator experiments. Specifically, here we used a 1 mA, 1 second boxcar pulse in a binaural-bipolar configuration. This led to a sensation of virtual roll which was matched to left and right turns only. By contrast, Cevette et al. (2012) used GVS in a four-pole

configuration at 1.5-2.5 mA intensity, matching GVS and visual cues for self-motion by providing virtual rotation in three axes. In addition, Reed-Jones et al. (2007) used a binaural-bipolar GVS configuration at each participants' threshold (0.6-1.25 mA). Thus, it may be possible that some degree of tailoring the vestibular stimulus to the participants' perception is necessary, rather than a standard GVS waveform across all participants.

During natural motion, the vestibular system encodes angular acceleration in roll, pitch, and yaw via the semicircular canals, and linear acceleration from both translation and gravity via the otolith organs. Importantly, the vestibular system responds quickly and dynamically to head acceleration and rotation, providing a comprehensive account of complex 3D motion in space (Carriot, Jamali, Chacron, & Cullen, 2014; Cullen, 2019). At present, the exact equivalence between the GVS-induced virtual rotation vector and real rotation elicited by natural motion is not entirely understood. It seems likely that GVS activates both otolith and semicircular canal afferents, although debate is ongoing (Cohen, Yakushin, & Holstein, 2012; Curthoys & MacDougall, 2012; Kim, 2013). Anodal currents decrease vestibular nerve firing rates, while cathodal currents increase them (Goldberg, Smith, & Fernández, 1984). Participants subsequently experience a polarity-dependent virtual roll rotation towards the cathode (Cathers et al., 2005; Fitzpatrick & Day, 2004). Accordingly, postural reflexes are elicited by GVS in standing participants, with an initial rapid tilt towards the anode, followed by an ongoing sustained tilt in the same direction (Cathers et al., 2005; Wardman, Day, & Fitzpatrick, 2003; Wardman, Taylor, & Fitzpatrick, 2003). While these postural reflexes have been well-studied, quantification of the perceived rotation vector has not been fully documented. Moreover, while more complex GVS configurations have been reported to elicit rotation in pitch and yaw

(i.e., four-pole GVS Aoyama, Iizuka, Ando, & Maeda, 2015; Cevette et al., 2012), the majority of studies have focused on the typically used binaural-bipolar configuration, resulting in sensations of roll. Thus, these unknowns may make full integration of GVS and VR difficult. As such, further research is necessary to identify exact parameters of artificial vestibular stimulation which could closely match visual motion in VR.

Secondly, previous studies differ between control conditions used to explore the effect of GVS on simulator sickness. Specifically, Reed-Jones et al. (2007) investigated the effect of GVS relative to a control in which no GVS was administered. In this case, a non-specific alerting effect of stimulation driving the reduction in simulator sickness scores cannot be excluded. In addition, Cevette et al. (2012) used a low-intensity (1 mA) constant GVS stimulation as a control, compared to higher intensity (1.5-2.5 mA) GVS pulses in the experimental condition. Previous research suggests that the perceived rotation from binaural-bipolar constant GVS declines to zero after approximately 100 seconds (St George, Day, & Fitzpatrick, 2011). Thus, it is not possible to rule out that participants habituated to the control stimulus across the 20-minute simulator exposure, while ongoing non-specific effects could account for the reduction in symptoms in the experimental condition. Alternatively, while the authors claimed that constant 1 mA GVS induced no sensations of motion, previous studies report postural responses to GVS as low as 0.3 mA (Wardman, Day, et al., 2003). As such, it is possible that participants in this control condition experienced sensations of motion which were not coupled with the visual motion presented in the flight simulator, worsening symptoms of simulator sickness relative to an alternative control in which vestibular cues are completely absent. Overall, these possibilities make interpretation of the effects of GVS difficult. In the present study, a sham-control was used whereby stimulation was applied to the base of the neck. Thus, participants

experienced cutaneous sensations in the absence of sensations of motion. Importantly, this stimulation was applied at the same intensity and at the same points as the GVS, ruling out the possibility of habituation. We found no difference between Sham and GVS, and as such we cannot exclude that a non-specific alerting effect could account for previous results.

Thirdly, previous studies investigating simulator sickness and GVS used scenarios in which participants actively controlled their motion through the environment (Cevette et al., 2012; Reed-Jones et al., 2007). By contrast, we used a passive scenario, in which the participants were moved through the virtual world on a set trajectory. Previous research suggests that passive VR scenarios tend to elicit greater levels of cybersickness than active ones (Sharples, Cobb, Moody, & Wilson, 2008; Stanney & Hash, 1998). This may be due to an increased level of predictability of sensory outcomes (Reason, 1978; Stanney & Hash, 1998). It may therefore be possible that artificial vestibular stimulation reduces cybersickness only when sensory outcomes are predicted by active control of the virtual environment. Future research could therefore directly compare the efficacy of GVS versus Sham stimulation in active versus passive VR scenarios.

Finally, we used a repeated measures design, whereby participants completed the study with both GVS and Sham stimulation. While this is in line with previous studies exploring GVS and simulator sickness (Reed-Jones et al., 2007), and although we took care to prevent carry-over effects by separating the sessions by one week, the SSQ results suggest that order was a significant factor in cybersickness scores. Specifically, participants experienced reduced cybersickness in their second session, irrespective of the stimulation type. This finding is in accordance with previous research suggesting that cybersickness tends to reduce with increasing exposure to VR

as participants become more familiar with the environment (Bailenson & Yee, 2006; Duh, Harm, & Parker, 2002; Howarth & Hodder, 2008). Thus, future research could consider using a between-subjects design (Cevette et al., 2012) to explore the role of GVS in reducing levels of cybersickness.

As well as a lack of change in SSQ scores, we also found no differences in VEMPs asymmetry ratios or amplitudes. These results do not accord with our previous findings, in which we found greater VEMPs asymmetry ratios following exposure to linear vection in VR (Gallagher et al., 2019). However, key differences between the previous findings and the present study are apparent. Specifically, while Gallagher et al. (2019) used very short duration vection, in the present study we used a 10-minute VR scenario. It is therefore feasible that changes in VEMPs vary across time, accounting for the contrasting findings in the present study. In particular, there may be more profound changes during the initial exposure to the VR-induced visuo-vestibular conflict, before stabilising after greater exposure. The time-course of VR aftereffects has not been extensively studied, and therefore further research should consider differences between VR duration on vestibular processing. Moreover, in the present study we used a detailed virtual environment, with both linear and rotational components as the virtual car navigated the environment. By contrast, previous findings isolated vection to a single axis of motion, without using a detailed VR scenario (Gallagher et al., 2019). Thus, it is possible that the differences in visual stimulus type could account for the lack of significant findings in the present study. Finally, we note that in the present study VEMPs data was overall highly variable across the sample of participants, despite using well-controlled and standardised methods. We therefore cannot rule out that this variability may also account for the lack of change according to exposure to our VR scenario.

Overall, in the present study we found no significant changes in cybersickness when participants engaged in a VR scenario with GVS. This contrasts with previous findings in simulator sickness (Cevette et al., 2012; Reed-Jones et al., 2007), however differences in stimulation protocols and VR scenarios could account for this divergence. Importantly, in the present study we used a sham-control which was as close to GVS as possible while excluding general vestibular activation and sensations of motion. Thus, it is not possible to rule out that general alerting effects account for previously reported reductions in simulator sickness. Finally, it is important to consider that the vestibular system signals complex, dynamic self-motion, and the equivalence between GVS and natural motion is not well understood. Uncovering the equivalent natural motion accounted for by GVS would therefore enable VR designers to more closely match the virtual rotation and visual motion in VR, potentially further improving the efficacy of GVS on cybersickness.

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Chapter 8:

Quantifying Virtual Self-Motion Sensations Induced by Artificial Vestibular Stimulation

Word count: 4,468

Abstract

The vestibular system provides a comprehensive estimate of self-motion in 3D space. Galvanic Vestibular Stimulation (GVS) is widely used to artificially stimulate the vestibular system. As well as activating a wide cortical network, GVS also elicits a clear virtual sensation of rotation on the roll axis. Postural responses to GVS have been clearly delineated, however quantifying the perceived virtual rotation vector has not been fully realised. Critically, the virtual self-motion triggered by GVS could prove useful in contexts such as sensory substitution in vestibular patients or in designing immersive Virtual Reality environments. Here we aimed to quantify the perceived virtual rotation vector elicited by GVS using a 3D turntable. We estimated that supine participants perceived a virtual roll rotation towards the cathode of approximately 2°/s velocity for 1 mA GVS and 6°/s velocity for 2.5 mA GVS. Crucially, these estimates were based purely on perceptual judgements, in the absence of any motor or postural responses to the stimulation and in a head orientation where the GVS-induced roll sensation did not interact with the perceived direction of gravity. The observed values were also stable across repetitions. This is an important step towards applications of GVS in sensory substitution or Virtual Reality contexts.

Introduction

Moving through the environment elicits a host of multisensory information regarding the location of the body in 3D space. The visual system detects optic flow from the external world, proprioception signals the position of the body, and vestibular signals provide information regarding acceleration (Greenlee et al., 2016). These vestibular signals are particularly important for self-motion (Britten, 2008; DeAngelis & Angelaki, 2012; Dichgans & Brandt, 1978). The vestibular organs are located inside the inner ears and are comprised of different structures: three orthogonal semicircular canals (anterior, posterior and lateral) which detect angular rotations of the head in roll, pitch and yaw, and two otolith organs (utricle and saccule) which detect linear acceleration from both translation and gravity. The integration of signals from these vestibular receptors provide us with a comprehensive representation of the motion of our head in 3D space (Cullen, 2019; Glover, 2004).

A widely used and well-controlled method of artificially stimulating the vestibular system is Galvanic Vestibular Stimulation (GVS). Electrodes are placed on the mastoids, stimulating the vestibular nerve (Curthoys & MacDougall, 2012; Kim, 2013; Stephan et al., 2005). Although debate is still ongoing, it seems likely that GVS stimulates both semicircular canal and otolith afferents (Curthoys & MacDougall, 2012; Kim, 2013; Kwan, Forbes, Mitchell, Blouin, & Cullen, 2019; Stephan et al., 2005, but see Cohen, Yakushin, & Holstein, 2012 for contrasting findings). Typically, a bipolar-binaural configuration is used, with anodal currents decreasing vestibular nerve firing rates and cathodal currents increasing them (Goldberg, Smith, & Fernández, 1984). A wide bilateral cortical network has been shown to be activated by GVS, including the insula, parietal operculum, midcingulate cortex, and somatosensory cortices (Lobel, Kleine, Bihan, Leroy-Willig, & Berthoz, 1998; Lopez,

Blanke, & Mast, 2012; Zu Eulenburg, Caspers, Roski, & Eickhoff, 2012). In the past decades, GVS has been largely used to investigate the role of the vestibular system in a range of perceptual and cognitive tasks, including body representation, decision making and visual spatial attention (Ferrè, Berlot, & Haggard, 2015; Lepecq, 2006; Mast, 2010; Volkening et al., 2014).

Postural responses are triggered by GVS when subjects are standing (Cathers, Day, & Fitzpatrick, 2005; Fitzpatrick & Day, 2004; Wardman, Taylor, & Fitzpatrick, 2003). Wardman, Day and Fitzpatrick (2003) administered 0.3 mA and 0.5 mA of GVS to participants standing upright and measured the postural response during and after eight seconds of square wave stimulation. An initial rapid response towards the anode was seen in the first second of stimulation. This initial step was followed by a continuous movement towards the anode until the point at which GVS was stopped, and participants returned gradually towards the start position. Thus, the GVS postural response was summed as an initial rapid step followed by a constant-velocity ramp towards the anode. Importantly, stimulation intensity seems to be crucial: GVS at 0.5 mA resulted in a greater displacement than stimulation at 0.3 mA (Wardman, Day, et al., 2003). Interestingly, modifying the position of the head relative to the body can change the postural response elicited by GVS. Cathers et al. (2005) administered 2 mA of GVS when the head was turned over the shoulder and either upright or pitched downwards. By adopting these head postures, the axis of rotation shifts from a sensation of roll to pitch or yaw respectively in head coordinates. Crucially, while a sensation of pitch requires significant postural adjustments to maintain balance, the sensation in yaw does not. Accordingly, when the head was upright, a large sway response towards the anode was seen at GVS onset, with a return to the original posture on GVS offset. By contrast, when the head was positioned downwards, only small

transient responses towards the cathode were seen. Taken together, these results suggest that vestibular inputs from GVS are integrated in function of head position, resulting in altered sensations of motion and appropriate postural responses (Cathers et al., 2005). In addition, greater postural displacements are seen with higher GVS intensities (Wardman, Day et al., 2003).

But what is the *perceptual* sensation associated with GVS? It has been consistently reported that binaural bipolar GVS results in a polarity-dependent virtual roll-rotation vector, where the individual perceives a sense of roll rotation towards the cathode (Cathers et al., 2005; Fitzpatrick & Day, 2004; Fitzpatrick, Marsden, Lord, & Day, 2002). Day and Fitzpatrick (2005) and Fitzpatrick and Day (2004) proposed that the virtual rotation vector induced by GVS arises as a result of changes in vestibular afferent firing rates mimicking a real motion of the head in space. Real head motion stimulates one or more pairs of semicircular canals, generating opposite changes in the firing rates of the respective vestibular afferents. The change in firing rate corresponds to the magnitude of the rotation vector perpendicular to each semicircular canal plane. The signals from the semicircular canals can therefore be vector summed to provide a net rotation vector in skull-fixed coordinates. When the system is stimulated using GVS, the semicircular canal vector depends on GVS intensity, with greater perceived rotation with higher GVS intensities (Day & Fitzpatrick, 2005). The net GVS-evoked virtual rotation vector is then computed as a vector dot product between gravitational cues regarding the location of the head in space and the semicircular canal vector sum, resulting in a rotation axis estimated to pass 18.8° below Reid's plane (Day & Fitzpatrick, 2005; Fitzpatrick & Day, 2004). Given the angle of the GVS rotation axis, perceived rotation reverses direction when the head is pitched backwards or forwards (Day & Fitzpatrick, 2005). Crucially, the previously described postural responses

appear to accord with these predictions (Cathers et al., 2005; Wardman, Day, et al., 2003).

While neuroimaging and postural effects of GVS have been extensively studied, a detailed quantification of the perceived virtual rotation vector has not yet been achieved. Quantifying the self-motion sensation elicited by GVS is however an essential step not only for the theoretical understanding of this technique, but also for its potential applications. Specifically, GVS could be used as a sensory substitution method in patients with bilateral vestibular loss who may benefit from the stimulation to restore lost vestibular function (Peterka, 2012; Wuehr, Decker, & Schniepp, 2017). In addition, GVS may also be implemented to provide additional vestibular cues in virtual reality (VR) (Cevette et al., 2012; Maeda, Ando, & Sugimoto, 2005). During VR, vision signals that the user is moving through the environment, while vestibular cues signal that the user is stationary. This visuo-vestibular conflict can result in symptoms of nausea, disorientation, fatigue, and oculomotor disturbance, known as *cybersickness* (Keshavarz & Hecht, 2011; Rebenitsch & Owen, 2016). Combining artificial vestibular cues from GVS with VR could therefore prevent visuo-vestibular conflict and reduce adverse symptoms during VR use (Cevette et al., 2012; Reed-Jones, Reed-Jones, Trick, & Vallis, 2007). However, precise estimates of the natural equivalent motion of GVS need to be described for these applications to be effective. Moreover, it is unknown whether the virtual rotation sensation evoked by GVS is stable across time and multiple exposures. Interestingly, Ertl, Klimek, Boegle, Stephan and Dieterich (2018) found that GVS detection thresholds were similar across repeated sessions on different days, which might suggest that the sensations evoked by GVS are similar across different exposures. Whether the virtual rotation percept itself remains robust across time has not been investigated.

Here we aimed to quantify the natural equivalent perceived motion of binaural-bipolar boxcar (i.e., square wave) GVS. Participants were physically rotated on a 3D-turntable while GVS was applied, eliciting virtual rotation in the opposite direction to the physical rotation. Using controlled psychophysical methods, we estimated the point of equivalence between natural motion and GVS sensations and investigated its stability over time. Given previous research (Cathers et al., 2005; Day & Fitzpatrick, 2005; Wardman, Day, et al., 2003), we expected participants to experience a sensation of roll rotation towards the cathode, and we expected no significant difference between repeated sessions.

Methods

Ethics

The experimental protocol was approved by the Canton of Zurich ethics committee. The study was conducted in line with the Declaration of Helsinki. Written informed consent was obtained from participants prior to commencing the study.

Participants

Eight participants (three female, mean age = 34.38, SD = 12.34) completed the study, including authors M.G., G.B., F.R. and C.B.. Six participants were right-handed according to their Edinburgh Handedness Inventory (Oldfield, 1971) scores, while the remaining two were left-handed. Exclusion criteria were any history of neurological, psychiatric, or vestibular conditions, epilepsy or family history of epilepsy. All participants' data was included in the final analysis, resulting in a total sample size of eight participants.

Procedure

After completing informed consent procedures, participants were given task instructions. Participants were secured on a human 3D-Turntable at University of Zurich, Department of Neurology, positioned so that the centre of the head was at the intersection of the three rotation axes. A response bar and button were fixed just in front of the participants' hands. At the beginning of the experiment, the turntable was rotated such that the participants were supine during the experiment. This posture was chosen to minimise confounds of position change with respect to gravity during the trials. The experiment consisted of trials where physical motion stimuli were delivered by rotating the turntable clockwise and anticlockwise around an Earth vertical axis passing through the centre of the head. These stimuli elicited a roll sensation to the left and right respectively.

The motion profile of the turntable in each trial consisted of a "velocity step" followed by a ramp to counteract semicircular canal adaptation, and to mimic GVS sensations as closely as possible (Wardman, Day, et al., 2003). For the steps, the 3D-Turntable moved with an initial acceleration of $20^{\circ}/s^2$ until the trial velocity was achieved. The turntable continued to accelerate steadily at $1^{\circ}/s$ (ramp) until the end of the trial (i.e. once a response had been provided or after 5 seconds). Once the turntable stopped rotating, the participants were instructed to commence the next trial only once all sensations of rotation had subsided. A minimum break of two seconds was enforced between trials. The experiment was conducted in darkness, so no visual cues for rotation were available. In addition, padding was placed around the participants' legs, to minimise somatosensory cues during rotation.

GVS was administered by a commercial stimulator (Good Vibrations Engineering Ltd., Nobleton, ON, Canada). Electrodes measuring approximately $4cm^2$

were coated with NaCl electrode gel and placed on the mastoids (GVS) or on the base of the neck (Sham), approximately 5 cm below the ear. Left-anodal/right-cathodal (L-GVS) stimulation was applied for clockwise trials while right-anodal/left-cathodal (R-GVS) stimulation was applied for anticlockwise trials. As GVS induces a sensation of rotation towards the cathodal side, the stimulation therefore induced a rotation sensation in the opposite direction to the rotation of the 3D-Turntable (Figure 1A). GVS was administered at two different intensities. Both rotation staircases included both polarities of GVS in a boxcar waveform of 5.5 seconds duration, with separate blocks of 1 mA and 2.5 mA.

A sham stimulation condition was also used to control for non-specific sensations, in which stimulation was applied via the electrodes placed on the base of the neck. This stimulation therefore elicits similar cutaneous sensations as GVS without subsequent activation of the vestibular nerve, resulting in no sensations of rotation. This type of stimulation may also control for the participants' idea that an unusual stimulation is occurring, accounting for cognitive factors. Sham stimulation was 2.5 mA, 5.5 seconds duration, and also delivered in left-anodal/right-cathodal (L-Sham) and right-anodal/left-cathodal (R-Sham) polarities according to the direction of 3D-Turntable rotation. Sham at 2.5 mA was chosen to control for the highest intensity of GVS used in the active stimulation conditions.

Participants pressed a button to start each trial. Once the button was pressed, the turntable started to move according to the velocity selected by the QUEST+ algorithm (Watson, 2017) and the GVS/Sham stimulation was triggered. Two seconds after the turntable had reached the selected velocity, a beep was sounded to indicate that the participants should report their perceived direction of rotation by rotating the bar clockwise or anticlockwise. A minimum of 50 trials were used for both clockwise

and anticlockwise staircases. The staircases were interleaved within each block, resulting in a minimum of 100 trials per block.

Participants completed four sessions on different days, separated by four days to one week. In each session, all participants first completed the baseline block with sham stimulation, while 1 mA and 2.5 mA GVS conditions were counterbalanced across participants and sessions such that each participant completed each order (i.e., 1 mA or 2.5 mA first) twice across their four sessions. A practice block of 10 trials was completed by all participants before the first session.

Data Analysis

Data from each staircase were fitted with cumulative normal psychometric functions in MATLAB r2017a, providing the threshold (or point of subjective equality, PSE, i.e., the velocity in degrees at which GVS and turntable rotation sensations were cancelled) and slope (which indicated the participants' precision). Positive PSE values corresponded to an anticlockwise rotation of the head, while negative values corresponded to a clockwise rotation of the head. Higher slope values indicated lower precision.

PSEs and slopes were calculated for each participant, GVS amplitude, and session. Data which was ±2.5 median absolute deviations from each participants' median PSE and/or slope was excluded. PSEs and slopes were fitted with linear mixed effects models in R with lme4 (Bates, Mächler, Bolker, & Walker, 2015; R Core Team, 2017). GVS Amplitude and Sessions were fixed factors and Participant was a random factor. Likelihood ratio tests with and without the fixed effects in question were used to obtain *p* values.
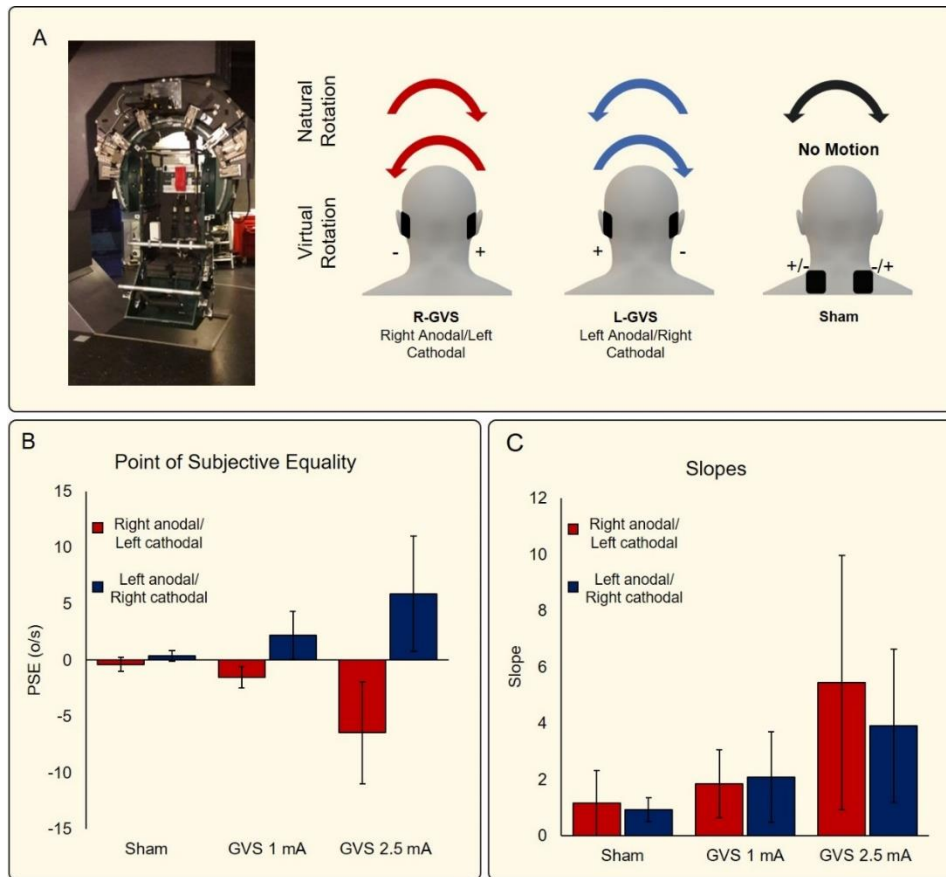
Figure 1. Experiment set up and results. A) 3D turntable and GVS configuration. GVS always elicited virtual rotation in the opposite direction to the physical rotation. B) PSE results across GVS amplitudes and polarities. C) Slopes across GVS amplitudes and polarities.

Results

Point of Subjective Equality

Overall means and standard errors of PSE values between Sham stimulation and both GVS amplitudes can be seen in Figure 1B. Individual LGVS data points can be seen in Appendix 4, Figure A20, while individual RGVS data points can be seen in Appendix 4, Figure A21. All raw PSE data are available in Appendix 5, Table A14. As expected, descriptive statistics showed that Sham stimulation elicited no motion sensations, with values close to 0. Importantly, GVS elicited a sensation of roll rotation towards the side of the cathode, as expected from previous research (Cathers et al.,

2005; Fitzpatrick et al., 2002). Moreover, the PSE increased with higher amplitudes of GVS, such that higher velocities of natural vestibular stimulation were required to cancel the GVS sensations.

The best-fitting linear mixed effects model for PSE values during L-GVS was one including only GVS as a fixed factor (AIC = 363.59; $X^2(2) = 45.09$, $p < .001$). The intercept for this model (i.e., Sham stimulation) was 0.37°/s ± 0.86 (SE). 1 mA GVS increased the PSE by 1.63°/s ± 0.75 (SE) from Sham values on average, while 2.5 mA GVS increased the PSE from Sham on average by 5.81 °/s ± 0.73 (SE). The random effect of Participant had a variance of 3.75.

Similar effects were seen on PSE values for R-GVS (AIC = 316.50; $X^2(2) = 54.86$, $p < .001$). For this polarity, the intercept was -0.32°/s ± 0.66 (SE), with 1 mA GVS decreasing PSE estimates from Sham values on average by -1.13 °/s ± 0.69 (SE), and 2.5 mA decreased the PSE from Sham on average by -6.31 °/s ± 0.69 (SE). The random effect of Participant had a variance of 1.64.

Neither model including Session as a fixed factor was significantly better at predicting thresholds than the GVS only model ($p > .05$). PSEs for L-GVS and R-GVS across each session can be seen in Table 1. Summary statistics for each participant can be seen in Table 2.

Table 1. Mean (SD) PSEs across GVS amplitudes, polarities, and experiment sessions.

Session	Sham		1 mA		2.5 mA	
	L-GVS	R-GVS	L-GVS	R-GVS	L-GVS	R-GVS
1	0.73 (0.90)	-0.47 (0.66)	2.83 (3.06)	-1.04 (0.78)	7.45 (6.96)	-7.41 (3.86)
2	0.43 (0.43)	-0.03 (0.29)	2.82 (2.91)	-1.58 (0.74)	6.02 (5.52)	-5.15 (4.81)
3	0.09 (0.35)	-0.32 (0.41)	1.83 (1.20)	-1.47 (1.07)	4.67 (2.94)	-7.73 (5.67)
4	0.31 (0.16)	-0.53 (0.88)	1.42 (0.97)	-1.88 (1.13)	4.73 (4.13)	-4.33 (3.46)

Table 2. Mean (SD) PSEs averaged across sessions for each Participant, GVS Polarity and Amplitude.

	Sham		1mA		2.5mA	
Participant	L-GVS	R-GVS	L-GVS	R-GVS	L-GVS	R-GVS
1	0.30 (0.28)	0.13 (0.21)	2.95 (0.49)	-0.90 (0.14)	5.85 (0.07)	-8.90 (1.13)
2	0.33 (0.12)	-0.27 (0.25)	0.85 (0.07)	-1.00 (0.17)	2.95 (1.48)	-4.90 (6.42)
3	0.0 (0.18)	-0.50 (0.14)	1.35 (0.42)	-2.10 (0.14)	3.53 (1.56)	-7.50 (0.57)
4	0.70 (0.26)	-1.95 (0.07)	3.00 (0.10)	-2.53 (0.15)	10.80 (0.10)	-10.25 (0.21)
5	0.20 (0.00)	0.07 (0.12)	1.40 (0.20)	-0.40 (0.14)	2.87 (1.21)	-2.30 (0.82)
6	0.43 (0.15)	0.05 (0.07)	0.85 (0.07)	-0.20 (0.10)	5.10 (1.61)	-4.20 (1.15)
7	0.23 (0.51)	-0.80 (0.26)	0.33 (0.25)	-2.30 (0.72)	2.83 (1.53)	-3.90 (0.98)
8	0.60 (1.28)	-0.30 (0.32)	5.43 (3.32)	-2.23 (0.51)	19.90 (4.53)	-13.57 (4.08)

Slopes

Means and standard errors for slopes between Sham and both GVS amplitudes can be seen in Figure 1C. Individual LGVS data points can be seen in Appendix 4, Figure A22, while individual RGVS data points can be seen in Appendix 4, Figure A23. All raw slope data are available in Appendix 5, Table A15. Descriptive statistics showed that precision decreased with GVS vs Sham stimulation, with decreased precision also with increasing amplitudes of GVS.

The best-fitting linear mixed effects model for slopes during L-GVS was one including only GVS as a fixed factor (AIC = 286.19; (X^2(2) = 37.34, p < .001). The intercept was 1.10°/s ± 0.49 (SE). 1 mA GVS increased slopes from Sham on average by 0.96 ± 0.44 (SE), while 2.5 mA GVS increased slopes from Sham by 3.00 ± 0.43 (SE) on average. The random effect of Participant had a variance of 1.14.

Similar effects were seen for R-GVS (AIC = 319.08; X^2(2) = 33.60, p < .001). For this polarity, the intercept was 1.00°/s ± 0.70 (SE). 1 mA GVS increased slopes from Sham on average by 0.74 ± 0.70 (SE), while 2.5 mA increased slopes from Sham by -4.42 ± 0.69 (SE) on average. The random effect of Participant had a variance of 1.99.

Neither model including Session as a fixed factor was significantly better at predicting slopes than the GVS only model ($p > .05$). Slopes for L-GVS and R-GVS across each session can be seen in Table 3. Summary statistics for each participant can be seen in Table 4.

Table 3. Mean (SD) Slopes across GVS amplitudes, polarities, and experiment sessions.

	Sham		1 mA		2.5 mA	
Session	**LGVS**	**RGVS**	**LGVS**	**RGVS**	**LGVS**	**RGVS**
1	1.20 (0.18)	1.59 (1.64)	2.65 (2.41)	2.26 (2.05)	4.96 (3.14)	4.50 (4.24)
2	1.22 (0.61)	1.33 (1.37)	2.48 (1.78)	2.03 (1.07)	3.57 (2.29)	6.50 (5.49)
3	0.73 (0.32)	0.75 (0.31)	1.67 (1.25)	1.65 (0.61)	3.57 (3.25)	6.53 (5.14)
4	0.69 (0.20)	0.95 (0.94)	1.40 (0.46)	1.55 (0.99)	2.88 (1.42)	3.30 (1.92)

Table 4. Mean (SD) Slopes averaged across sessions for each Participant, GVS Polarity and Amplitude.

	Sham		1mA		2.5mA	
Participant	**L-GVS**	**R-GVS**	**L-GVS**	**R-GVS**	**L-GVS**	**R-GVS**
1	0.95 (0.07)	0.83 (0.45)	3.20 (0.28)	1.50 (1.41)	7.80 (0.57)	4.90 (0.85)
2	1.10 (0.00)	0.57 (0.15)	1.20 (0.14)	2.23 (0.67)	3.33 (1.43)	15.07 (1.71)
3	0.75 (0.21)	0.65 (0.07)	1.48 (0.43)	1.45 (0.07)	1.63 (0.67)	2.25 (0.21)
4	1.27 (0.74)	3.05 (0.35)	1.47 (0.65)	1.50 (1.08)	1.90 (0.2)	2.35 (0.07)
5	0.50 (0.00)	0.47 (0.21)	2.37 (1.24)	0.65 (0.21)	8.07 (2.33)	3.58 (1.28)
6	0.83 (0.46)	0.35 (0.07)	0.65 (0.07)	0.83 (0.49)	2.20 (0.70)	2.40 (0.50)
7	0.90 (0.78)	0.90 (0.26)	1.30 (0.61)	2.63 (0.35)	2.90 (1.15)	2.90 (2.38)
8	1.00 (0.36)	2.35 (1.93)	4.13 (2.67)	3.27 (1.93)	7.40 (0.85)	8.40 (1.21)

Discussion

GVS has been widely used in research to investigate the role of vestibular afferents in perception and cognition (Ferrè et al., 2015; Lepecq, 2006; Mast, 2010; Volkening et al., 2014). As well as activating a wide vestibular cortical network (Lobel et al., 1998; Lopez et al., 2012; Zu Eulenburg et al., 2012), GVS also induces a virtual

roll-rotation sensation towards the cathode (Day & Fitzpatrick, 2005; Fitzpatrick & Day, 2004). Although previous research has quantified the postural responses elicited by GVS (Cathers et al., 2005; Wardman, Day, et al., 2003; Wardman, Taylor, et al., 2003), precise estimates of the *perceived* virtual rotation vector have not been fully described. In the present study we used a psychophysical protocol to find the point of equality between GVS-induced virtual rotation and natural rotation. In accordance with previous accounts (Cathers et al., 2005; Day & Fitzpatrick, 2005; Fitzpatrick & Day, 2004; Wardman, Taylor, et al., 2003), GVS at both 1 mA and 2.5 mA induced a virtual roll-rotation towards the cathode. Moreover, the virtual rotation vector increased with higher amplitudes of GVS. Crucially, we also demonstrated for the first time that the GVS-induced virtual rotation was stable across repeated exposures.

Previous research has investigated differences between the illusionary motion evoked by GVS and natural motion. Specifically, while perceived rotation from a 120 second 20°/s natural rotation declined with a time constant of 15.8s, perceived yaw rotation elicited from 120 seconds of 1.5 mA GVS took 103 seconds to decline (St George, Day, & Fitzpatrick, 2011). In addition, while sensitivity to direction increases as the frequency of real sinusoidal motion increases, the reverse is apparent for sinusoidal GVS (Peters, Rasman, Inglis, & Blouin, 2015). Finally, while perceived motion was always in phase of real motion, this was only the case at lower frequencies of GVS: at higher frequencies, an advance of the phase was seen (Peters et al., 2015). Despite these investigations, no studies have precisely estimated the equivalent natural motion sensation induced by GVS.

Here we found that GVS elicited a virtual roll rotation vector of approximately 2°/s velocity for 1 mA stimulation and 6°/s velocity for 2.5 mA stimulation. Thus, crucially, the velocity of the GVS virtual rotation vector increased in function of GVS

amplitude and was coherent with the direction of stimulation. This suggests that the percept induced by GVS does not increase simply as a result of increased arousal or attention to the stimulation. Rather, the virtual rotation vector occurs as a direct result of changes in afferent modulation. Accordingly, GVS can be used to induce reliable vestibular-driven illusory self-motion sensations in the absence of other sensory signals. Importantly, while previous studies have investigated the GVS virtual rotation vector indirectly through examining postural responses to the stimulation (Cathers et al., 2005; Fitzpatrick et al., 2002; Wardman, Day, et al., 2003; Wardman, Taylor, et al., 2003), in the present study we quantified a vestibular percept in the absence of motor responses.

Here we considered roll motion while participants were supine. This posture was chosen to avoid participants utilising additional postural cues with respect to gravity as the 3D turntable moved. The integration of both semicircular canal and otolith cues is vital for accurately estimating self-motion. In particular, inertial acceleration from both tilting the head relative to gravity and linear translation produces an identical response at the otoliths (Angelaki, McHenry, Dickman, Newlands, & Hess, 1999; Glasauer, 1992; Green & Angelaki, 2010). Thus, semicircular canal cues must be integrated with otolith cues to distinguish between tilt and translation. Accordingly, when the head is upright ongoing positional cues regarding the head's location with respect to gravity are available. However, when supine only dynamic angular acceleration cues can be used to estimate self-motion in the coronal plane of the body (Vimal, DiZio, & Lackner, 2017). Moreover, the perceived rotation vector varies in function of the position of the head with respect to gravity (Day & Fitzpatrick, 2005). As such, further research could therefore investigate whether similar equivalent velocities are elicited from rotation on other axes with

respect to gravity. Previous research investigating the postural effects of GVS have frequently applied stimulation while the head is tilted forwards, therefore eliciting a sensation of whole-body yaw, rather than head roll (Cathers et al., 2005; Day & Fitzpatrick, 2005). In this position, smaller postural responses are elicited, potentially due to lower demands for maintaining balance in comparison to a roll rotation of the head (Cathers et al., 2005). It may therefore be interesting to explore whether the velocity of the virtual rotation is similar or attenuated when participants perceive body yaw by tilting the head during GVS.

In the past few decades, several studies have considered the effect of GVS on behaviour (Ferrè et al., 2015; Lepecq, 2006; Mast, 2010; Volkening et al., 2014), and have clearly mapped postural and neuroimaging responses (Cathers et al., 2005; Day & Fitzpatrick, 2005; Lobel et al., 1998; Wardman, Taylor, et al., 2003). However, no studies have precisely described the perceived virtual rotation induced by the artificial vestibular stimulation. A description of the virtual rotation vector is necessary if GVS is to be used as sensory substitution, or to enhance VR experiences. Moreover, the reliability of the virtual rotation vector must also be understood if GVS is to be applied in these contexts. Here we found that GVS elicited a sensation of roll rotation towards the cathode, with the velocity increasing with higher intensities of stimulation. Importantly, the rotation vector was similar across repeated sessions, suggesting that the virtual roll vector was stable across time. Thus, these findings quantify the roll sensations of GVS for the first time, potentially proving useful for applied contexts, such as sensory substitution or VR.

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Chapter 9:

Critical Evaluation and Conclusion

In my PhD thesis, I have outlined a visuo-vestibular multisensory integration framework for sensory processing in Virtual Reality (VR) which aims to explain cybersickness. I have also systematically explored predictions derived from this framework. Importantly, my results demonstrated for the first time how both perceptual and physiological vestibular processing can be dramatically altered after only brief exposure to vection in VR. Specifically, I found that vestibular detection was significantly worse when participants were exposed to visuo-vestibular conflicts within a plane of motion (Chapter 4). Moreover, I found that the asymmetry of vestibular-evoked myogenic potentials was significantly increased after exposure to linear vection (Chapter 5). Thus, these findings implicate a dynamic vestibular re-weighting process following exposure to VR.

In addition, I have also outlined potential physiologically-driven interventions for the prevention of cybersickness. In Chapter 6 I described how tilting the body with respect to the gravitational vertical could reduce the reliability of vestibular cues, inducing vestibular down-weighting to reduce cybersickness. In Chapter 7, I evaluated an integrated Galvanic Vestibular Stimulation (GVS)+VR driving scenario, which may reduce cybersickness by artificially substituting vestibular cues for self-motion, preventing visuo-vestibular conflicts. However, a critical evaluation of this intervention identified the lack of a quantitative description of the GVS-induced motion sensation as a substantial barrier for implementation. Thus, in Chapter 8 I quantified, seemingly for the first time, the natural equivalent perceived virtual motion induced by GVS, optimising vestibular stimulation parameters for future VR implementations. Taken together, my theoretical framework and the results supporting

it provide a clear, systematic approach to adverse effects of VR exposure, and can guide future work in this area.

In the final chapter, I will critically evaluate the research presented in my thesis, focusing on the framework and findings in the context of alternative cybersickness theories and previous empirical findings, as well as outline implications of the work along with limitations and future directions.

The Visuo-Vestibular Framework in Context

Cybersickness can be regarded as a sub-type of motion sickness (Mazloumi Gavgani, Walker, Hodgson, & Nalivaiko, 2018). Accordingly, theories of cybersickness development are predominantly based on general theories of motion sickness (Bos, Bles, & Groen, 2008; Oman, 1988; Reason & Brand, 1975; Riccio & Stoffregen, 1991). The most widely-accepted explanation for motion sickness, and therefore cybersickness, is a *sensory conflict* account (Bos et al., 2008; Oman, 1988; Reason & Brand, 1975). Importantly, evidence supports the notion of sensory conflict as an underlying cause of cybersickness: when there is a discrepancy between visual and vestibular cues for self-motion, for example by including rotation on more than one axis, symptoms of motion sickness are exacerbated (Akiduki et al., 2003; Bonato, Bubka, & Palmisano, 2009; Keshavarz & Hecht, 2011).

The visuo-vestibular multisensory integration framework outlined in my thesis is also based on the idea that sensory conflict is an underlying cause of cybersickness. Specifically, in typical VR scenarios – i.e. a rollercoaster or a flight simulator – visual cues signal that the user is moving through a virtual environment, while vestibular cues signal that the user is stationary. Importantly, the mechanism for VR adaptation

proposed in this framework is based on general accounts of multisensory integration for self-motion (DeAngelis & Angelaki, 2012; Fetsch, Turner, DeAngelis, & Angelaki, 2009; Gu, Angelaki, & DeAngelis, 2008). Specifically, cues are weighted according to their reliability, with dynamic changes occurring in weighting if senses become unreliable (Fetsch et al., 2009). Evidence clearly describes that bimodal estimates are more precise than the unimodal estimates alone (Angelaki, Gu, & DeAngelis, 2011; DeAngelis & Angelaki, 2012). Moreover, multisensory integration reduces uncertainty regarding the source percept (Green & Angelaki, 2010; Knill & Pouget, 2004). Accordingly, during exposure to VR, vestibular cues are down-weighted such that information regarding self-motion is predominantly extracted from visual cues. When vestibular cues are once again present after VR exposure, for example by physically moving through the real world, a second period of re-weighting occurs where vestibular cues are then up-weighted. This *dynamic re-weighting* of vestibular cues can therefore account for adaptation to VR, as well as VR after-effects (Gallagher & Ferrè, 2018).

Given the large discrepancy between visual and vestibular cues in VR, one might question whether multisensory integration occurs at all, which may preclude a dynamic re-weighting of vestibular inputs. However, multisensory integration processes have been found to occur even when large discrepancies between visual and vestibular cues are present (Butler, Campos, & Bülthoff, 2014; Kaliuzhna, Prsa, Gale, Lee, & Blanke, 2015). Specifically, visual and vestibular information appears to be optimally integrated despite differences in velocity profiles between the cues (Butler et al., 2014), or when visual and vestibular self-motion cues are presented on different axes (Kaliuzhna et al., 2015). The exact limits of the optimal integration process for visual and vestibular cues require further exploration, however it is possible that

conflicting stimuli are integrated when other properties of the stimuli, such as onset or duration, are correlated (Kaliuzhna et al., 2015). Thus, it is likely that a similar process occurs in VR, despite the discrepancies between visual and vestibular cues. The changes in vestibular processing reported in Chapters 4 and 5 may support a change in vestibular weighting following VR, however further assessment following exposure to more realistic VR scenarios might be needed in order to confirm this hypothesis.

How might the visuo-vestibular multisensory integration framework compare to other theories of cybersickness? In their neural mismatch theory Reason and Brand (1975) propose that motion sickness arises when incoming sensory inputs from self-motion do not match with stored predicted patterns of sensory cues, i.e., engrams, generating a mismatch signal. The mismatch signal is dependent on the number of sensory channels in conflict, the extent of the discrepancy between the channels, and the amount of previous exposure to the conflicting stimuli (Reason, 1978; Reason & Brand, 1975). Adaptation to motion sickness is achieved when new stored predictions can be formed, avoiding generation of mismatch signals. While extensive evidence suggests that sensory conflict is indeed a likely underlying mechanism for motion sickness (Akiduki et al., 2003; Bonato et al., 2009; Cohen, Dai, Yakushin, & Cho, 2019; Keshavarz & Hecht, 2011), this neural mismatch theory has been criticised for lacking a clear physiological basis, and cannot account for factors such as individual differences in motion sickness susceptibility, or why after-effects of VR arise (Davis, Nesbitt, & Nalivaiko, 2014; Oman, 1988; Warwick-Evans & Beaumont, 1995).

By contrast, the framework outlined in my thesis is based on established mechanisms of multisensory integration, outlining a potential physiological mechanism for VR adaptation (Ernst & Banks, 2002; Fetsch, DeAngelis, & Angelaki, 2013). Specifically, sensory conflicts arise from discrepancies between different

online sensory inputs, rather than between incoming sensory cues and stored engrams. Adaptation to the sensory conflict therefore requires dynamic re-weighting of visual and vestibular cues for self-motion, as opposed to storage of new engrams. Importantly, our framework can potentially account for factors not explained by Reason and Brand's (1975) neural mismatch theory (Angelaki, 2014; Ernst & Banks, 2002; Fetsch et al., 2009). Specifically, individual differences in cybersickness susceptibility may relate to individual differences in sensory integration processes, such as being influenced by visual dependence (Arshad et al., 2019; Cian, Ohlmann, Ceyte, Gresty, & Golding, 2011) or vestibular capture (Clément & Reschke, 2018; Fowler, Sweet, & Steffel, 2014; Neupane, Gururaj, & Sinha, 2018). In addition, after-effects may be accounted for by the dynamic re-weighting of vestibular cues following VR exposure.

An alternative account for motion sickness has been described considering vertical conflict (Bles, Bos, De Graaf, Groen, & Wertheim, 1998; Bos et al., 2008). According to this account, motion sickness arises not from general conflicts between sensory modalities, but specifically from conflicts in the predicted and sensed subjective vertical. Accordingly, levels of motion sickness correlate with the degree of tilt away from the vertical during off-vertical axis rotation (Dai, Sofroniou, Kunin, Raphan, & Cohen, 2010), and cybersickness is reduced when a stable visual reference for verticality is provided in VR (Chang et al., 2013; Han et al., 2011). Interestingly, the multisensory integration framework may accord with vertical conflict theory to some degree. Perception of the subjective vertical is constructed from visual and vestibular cues for gravity (Alberts, de Brouwer, Selen, & Medendorp, 2016; Alberts, Selen, et al., 2016; Harris, Jenkin, Dyde, & Jenkin, 2011). Thus, it is feasible that a discrepancy between these cues during VR exposure can lead to cybersickness. For

instance, a VR application presenting a visual viewpoint that the user is upside-down or tilted relative to gravity may be in disagreement with otolith cues suggesting that the user is upright. Accordingly, the sensory re-weighting process may down-weight vestibular cues for verticality and up-weight visual cues, as may be the case when the visual and vestibular cues for self-motion perception are in conflict. However, while this may be one possibility, the visuo-vestibular multisensory integration framework suggests that *any* discrepancy between visual and vestibular self-motion cues can lead to cybersickness, even in the absence of a conflict between cues for verticality. Moreover, many VR applications do not necessarily alter the subjective vertical, yet nonetheless they still produce cybersickness (Golding, 2016) – the integrated GVS+VR simulator presented in Chapter 7 for instance produced symptoms of cybersickness despite presenting a VR scenario in which the visual vertical aligned with the participants' body axis. Accordingly, the mechanism for cybersickness may be more general than that proposed by Bles et al. (1998) and Bos et al. (2008).

A final theory of cybersickness focuses on postural instability (Riccio & Stoffregen, 1991). According to this theory, motion sickness arises not as a result of sensory conflict, but rather occurs when individuals are exposed to situations in which they cannot precisely control their posture. Interestingly, increased postural sway may precede symptoms of cybersickness, and individuals who display greater postural instability may be more susceptible to motion sickness and cybersickness (Smart, Stoffregen, & Benoit, 2002; Stoffregen, Hettinger, Haas, Roe, & Smart, 2000; Stoffregen & Smart, 1998; Weech, Varghese, & Barnett-Cowan, 2018). However, recent empirical findings suggest that cybersickness may occur in the absence of postural instability, with changes in posture described selectively after occurrence of cybersickness symptoms (Dennison & D'Zmura, 2017, 2018). Moreover, situations in

which there are very few postural demands – i.e., when lying down or passively restrained – may entail similar levels of sickness as situations with greater postural demands (Warwick-Evans & Beaumont, 1995). Thus, while it seems that postural instability may have a role in cybersickness, the causal relationship may be still unclear. Importantly, similarly to the theories described above, the postural instability account cannot explain after-effects of VR. Like self-motion, postural control is also based on vestibular-multisensory integration (Chiba, Takakusaki, Ota, Yozu, & Haga, 2016; Oie, Kiemel, & Jeka, 2002; Schmuckler & Tang, 2019). Thus, the relationship between postural instability and cybersickness may reflect shared underlying mechanisms. However, further research investigating dynamic re-weighting for postural control and self-motion in VR is necessary to further uncover the link between the two concepts.

The visuo-vestibular multisensory integration framework I propose in my thesis provides a model for understanding cybersickness, VR adaptation, and VR-induced after-effects. Our framework extends sensory conflict theories of motion sickness by a mechanism-driven physiological approach, and accounts for factors that have been largely unexplained by previous theoretical models, such as individual differences and VR after-effects. The framework is founded on a reliability-based optimal integration model, however further assessments of vestibular re-weighting following VR would be an important step for future work. Overall, the framework can guide future research exploring cybersickness and VR aftereffects.

VR After-Effects: A Consequence of Vestibular Modulation?

At present, a full and systematic exploration of VR induced after-effects has not yet been conducted, which has led to a gap in our understanding. Previous research has reported increased disorientation (Stanney & Kennedy, 1998), poorer proprioceptive coordination (Harm, Taylor, Reschke, Somers, & Bloomberg, 2008; Stanney, Kennedy, Drexler, & Harm, 1999), and changes in vestibulo-ocular reflexes (Di Girolamo et al., 2001) following exposure to VR. However, the precise mechanism for these commonly described effects is unclear. The multisensory integration framework outlined in my thesis suggests that after-effects may arise due to the dynamic changes in re-weighting following exposure to VR. This is evidenced by the changes in physiological and perceptual vestibular functioning I reported in Chapters 4 and 5.

In Chapter 4, I described how detection of artificial vestibular cues was significantly worse after a short exposure vection. Importantly, this modulation happened only when visual cues were congruent with the sensation induced by GVS. Accordingly, these findings suggest that the modulation in vestibular weighting after exposure to VR is specific to the plane of motion presented, rather than an overall down-weighting of vestibular cues, or other non-specific effects. The vestibular system comprises of otolith organs (utricle and saccule) which detect linear translation and gravity, and three semicircular canals (posterior, anterior, lateral) which detect rotations in roll, pitch, and yaw. As such, it may be possible that vestibular cues are *selectively* down-weighted according to the exact visuo-vestibular conflict induced by VR. However, as many commercially available VR scenarios tend to include complex motion scenarios, it is feasible that general down-weighting of vestibular cues is present during exposure to VR.

In Chapter 5, I also demonstrated a dramatic modulation of vestibular reflexes following exposure to linear vection in VR. Specifically, Vestibular-Evoked Myogenic Potential (VEMPs) amplitude on the left sternocleidomastoid muscle was significantly increased following exposure to linear vection, increasing the asymmetry of the response. Such changes in physiological processing occurred clearly below the participants' conscious awareness, and therefore potential biases influencing the effect of VR on vestibular processing are ruled out.

At first glance it may appear that a *decrease* in the ability to detect artificial vestibular stimulation reported in Chapter 4 may contradict with an *increase* in VEMPs amplitude reported in Chapter 5. However, it is possible that these two findings reflect different stages of the dynamic re-weighting process due to differences in the type of vestibular stimulation applied. Importantly, the visuo-vestibular multisensory integration framework posits that two re-weighting processes occur throughout VR exposure. Firstly, a down-weighting of vestibular cues may be present when the user is immersed in VR. This is due to the contradiction between visual cues, which suggest that the user is moving, and vestibular cues, which signal that the user is stationary. Secondly, an up-weighting of vestibular cues may be present when the user emerges from the VR environment and moves in the real world, where visual and vestibular cues are once again congruent. Accordingly, the decrease in vestibular detection may reflect the initial down-weighting during VR exposure, while the increase in VEMPs amplitude may reflect the up-weighting following VR exposure. Importantly, the low-intensity GVS used in the study of Chapter 4 provided ambiguous and difficult to detect vestibular cues, while the sound-evoked vestibular stimulation used to elicit VEMPs in Chapter 5 was a clear, above-threshold vestibular stimulus. Thus, these stimulation parameters may be proxies for the ambiguous vestibular cues in VR and

the coherent vestibular cues post-VR respectively. As such, if the detection task utilised clearer vestibular stimuli, one might predict that vestibular detection would be improved as vestibular cues were up-weighted, reflecting the second re-weighting stage. Further research is necessary to explore this possibility, and to understand how vestibular processing is altered over time.

Interestingly, the changes in vestibular processing reported here occurred after very brief exposures to isolated vection in VR. Thus, open questions remain regarding vestibular processing following exposure to longer duration and more realistic VR scenarios. Di Girolamo et al., (2001) reported decreases in vestibulo-ocular reflex gain following 20 minutes of exposure to an immersive VR game, suggesting a down-weighting of vestibular cues. However, it remains unclear how the findings presented in my thesis would change if participants were exposed to a similar VR scenario for extended lengths of time. Given the predictions of the visuo-vestibular multisensory integration framework, one might hypothesise that the changes in vestibular processing would become more pronounced with greater lengths of exposure as the individual adapts to the conflicting environment, and the vestibular cues are further down-weighted. Moreover, given the apparent specificity in vestibular down-weighting, the changes in vestibular processing may differ according to the planes of motion presented in a more realistic VR environment. For instance, a VR game with more linear translations than rotations may have greater impacts on vestibular cues for translation, leaving vestibular cues for rotation relatively unaffected. Previous research suggests that more realistic VR scenarios may entail greater levels of cybersickness as the visuo-vestibular conflict is more pronounced (Davis et al., 2014; Merhi, Faugloire, Flanagan, & Stoffregen, 2007). Thus, modulation of vestibular processing may also differ according to the realism of the virtual environment.

The vestibular system has widespread projections throughout the human brain (Lopez, Blanke, & Mast, 2012; Zu Eulenburg, Caspers, Roski, & Eickhoff, 2012). As such, it is implicated in a large range of perceptual, behavioural, and cognitive functions (Bigelow & Agrawal, 2015; Hitier, Besnard, & Smith, 2014; Mast, 2010). Accordingly, it is possible that changes in vestibular processing induced by visuo-vestibular conflict in VR may induce wider after-effects than those pertaining to vestibular processing directly. For instance, changes in proprioceptive coordination (Harm et al., 2008; Stanney et al., 1999) and increases in disorientation (Stanney & Kennedy, 1998) may relate to changes in vestibular processing, however further studies are necessary.

Here I demonstrated how vestibular processing could be significantly modulated by VR exposure, as predicted by the visuo-vestibular multisensory integration framework. While these findings provide clear preliminary evidence for vestibular changes in response to VR, further research is necessary. Specifically, investigations of the time-course of the effects and whether the effects are similar with more realistic VR scenarios is necessary. Furthermore, it may be useful to understand whether the changes in high and low-level vestibular processing can account for other reported VR after-effects.

Possible Interventions to Prevent Cybersickness

As well as predicting changes in vestibular processing following VR exposure, the visuo-vestibular multisensory integration framework also predicts at least two specific interventions for reducing cybersickness symptoms. Firstly, down-weighting of vestibular cues through either artificial vestibular stimulation or tilting the body

with respect to gravity may reduce cybersickness by reducing the saliency of the visuo-vestibular conflict. Previous research using Bone Conducted Vibration (which induces noise into the vestibular system) successfully reduced symptoms of cybersickness, suggesting that down-weighting vestibular cues could indeed be an effective measure (Weech, Moon, & Troje, 2018). However, as Bone Conducted Vibration requires additional stimulation technology, it may not be a solution which is widely adopted by all VR users. In my thesis, I explored an alternative method of vestibular down-weighting based on vestibular physiology. Specifically, I tilted participants with respect to the gravitational vertical. In this posture, vestibular signals for gravitational linear acceleration are no longer reliable, and subsequently individuals rely more on visual cues (Alberts, de Brouwer, et al., 2016; Alberts, Selen, et al., 2016; Vimal, DiZio, & Lackner, 2017; Ward, Bockisch, Caramia, Bertolini, & Tarnutzer, 2017). Importantly, this strategy does not require additional technology or any modification of the normal VR setup, conferring considerable advantages over alternative cybersickness prevention measures. While I found a numerical difference in levels of sickness, tilted participants did not experience a significant reduction in cybersickness symptoms relative to upright participants. It is possible that this was due to habituation to the tilted posture (Eron, Cohen, Raphan, & Yakushin, 2008; St George, Day, & Fitzpatrick, 2011), a lack of down-weighting of dynamic semicircular canal cues (Day & Fitzpatrick, 2005; Vimal et al., 2017), or simply ineffective selection of the ideal angle and axis of body tilt. Accordingly, at present there is limited evidence for the efficacy of this solution, and further research is necessary.

A second intervention to prevent cybersickness predicted by the visuo-vestibular integration framework is prevention of visuo-vestibular conflicts. This may be achieved by locomotion through the real world (Chance, Gaunet, Beall, & Loomis,

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1998; Llorach, Evans, & Blat, 2014, however see Peck, Fuchs, & Whitton, 2011, for contradictory findings), or by use of sensory substitution methods, such as artificial vestibular stimulation. Previous studies have suggested that application of Galvanic Vestibular Stimulation (GVS) is an effective method to prevent simulator sickness, a related form of motion sickness occurring in non-VR immersive simulators (Cevette et al., 2012; Reed-Jones, Reed-Jones, Trick, & Vallis, 2007). Coupling visual motion with vestibular signals driven by GVS means that the two sensory modalities both signal that the user is moving, preventing the occurrence of symptoms (Cevette et al., 2012; Reed-Jones et al., 2007). However, application of GVS has not yet been tested in VR simulators, nor have ideal parameters for stimulation been described. In my thesis, I created an integrated GVS+VR driving simulator. Low-intensity, short-duration GVS pulses were applied during left and right turns of the driving simulator. Again, numerical differences were observed, however no significant changes were found between GVS and Sham stimulation conditions, contrasting with previous findings (Cevette et al., 2012; Reed-Jones et al., 2007).

While several factors could account for this lack of difference, a critical issue is that the direct relation between GVS-induced illusory motion and natural motion is not known. Previous research has delineated postural responses and a potential virtual rotation vector induced by GVS (Cathers, Day, & Fitzpatrick, 2005; Day & Fitzpatrick, 2005; Fitzpatrick & Day, 2004; Peters, Rasman, Inglis, & Blouin, 2015; Wardman, Day, & Fitzpatrick, 2003; Wardman, Taylor, & Fitzpatrick, 2003), however a direct quantification of the pure vestibular *percept* itself has not yet been conducted. In Chapter 8, I investigated this percept for roll-rotation while supine, and found that 1 mA of GVS was equivalent to a roll rotation of approximately 2°/s towards the cathode, with an increase in velocity with higher amplitudes of stimulation. A supine

posture was investigated to rule out the influence of gravitational cues, however this method could also be used to investigate rotation on other axes, and with more complex forms of GVS (Aoyama, Iizuka, Ando, & Maeda, 2015). Thus, future research investigating GVS as a method of cybersickness reduction in VR could use the parameters found in this work to create more effective integrated GVS+VR scenarios.

In my thesis, I have focused on the dynamic re-weighting of vestibular signals for prevention of cybersickness, as opposed to the re-weighting of visual cues. This is based on the idea that 1) individuals without functioning vestibular labyrinths do not experience motion sickness (Cheung, Howard, & Money, 1991; Paillard et al., 2013), highlighting the key role of the vestibular system in motion sickness development, and 2) visual cues for self-motion are a highly salient and key signal for self-motion in VR, suggesting that a down-weighting of these cues is unlikely. However, it is also feasible that mechanisms for down-weighting of visual cues may also prevent cybersickness. Accordingly, closing the eyes during rotational movements in VR can reduce symptoms (Kemeny, George, Merienne, & Colombet, 2017), as can dynamically reducing the field of view (Fernandes & Feiner, 2016). However, reports on reducing visual inputs during other forms of motion sickness are mixed. For instance, while closing the eyes while immersed in a moving-base flight simulator dramatically reduced simulator sickness (Ishak, Bubka, & Bonato, 2018), levels of sickness were higher with the eyes closed versus open when participants were driven in a car (Wada & Yoshida, 2016). Accordingly, further evidence is required to assess the efficacy of visual versus vestibular down-weighting in VR. Moreover, reducing cybersickness by modifying visual inputs may come at the expense of limiting the sense of presence and immersion in the virtual environment.

In sum, here I outlined two methods of cybersickness prevention based on the visuo-vestibular multisensory integration framework. While I did not find significant reductions in cybersickness symptoms, further refinement of the techniques could potentially provide further avenues for cybersickness prevention. Importantly, I also described the natural equivalent of the perceived motion induced by GVS, which is likely to prove useful for those investigating the technology as sensory substitution in VR.

Implications

One significant implication of my thesis is a new framework for cybersickness based on multisensory integration. This framework makes specific predictions regarding cybersickness development, VR induced after-effects, and mechanisms for cybersickness prevention. Accordingly, here I showed dramatic changes in vestibular processing following brief exposure to VR, and outlined two methods of cybersickness prevention which could form the basis for new research. Moreover, the framework also predicts that individual differences in multisensory integration could predict individuals who are likely to develop cybersickness. While this was not directly tested in my thesis, this could form the basis for future exploration. Specifically, those who rely more on vestibular cues over visual cues and those whose re-weighting function is slow to respond to the sensory conflict may be more likely to experience symptoms. Extensive research suggests that individuals differ in their reliance on vestibular and visual cues, and that this may modulate motion sickness susceptibility and self-motion perception (Arshad et al., 2019; Fowler et al., 2014, however, see Buyuklu, Tarhan, & Ozluoglu, 2009, and Weech, Varghese, et al., 2018 for contrasting findings). Thus,

investigating individual differences in multisensory re-weighting could provide further answers to predicting cybersickness.

A second key implication of this thesis are the findings suggesting a modulation of vestibular processing after exposure to vection in VR. Our understanding of VR-induced after-effects is lacking, and at present no theories can fully account for why after-effects develop. Previous reports of after-effects include disorientation, poorer proprioception, and changes in vestibulo-ocular reflex gain. The studies presented here suggest that a down-weighting of vestibular cues following VR could underpin these after-effects, providing avenues for future work. Interestingly, previous research has shown modulation of visual sensitivity following exposure to vestibular stimulation (Edwards, O'Mahonys, Ibbotson, & Kohlhagen, 2010; Shirai & Ichihara, 2012, but see Holten & MacNeilage, 2018, for contrasting findings). However, the reverse modulation has not been previously demonstrated. Thus, the work presented here also further emphasises the link between visual and vestibular cues for self-motion perception.

Finally, it is possible that the visuo-vestibular multisensory integration framework could be applied to forms of motion sickness beyond cybersickness. In particular, motion sickness arising from conflicts between visual and vestibular cues may be explained by this framework. For instance, space adaptation syndrome, a form of motion sickness arising from exposure to microgravity, may be caused by unreliable otolith cues conflicting with visual cues for orientation (Bertolini & Straumann, 2016; Lackner, 2014). Accordingly, a similar dynamic re-weighting process may lead to adaptation to the microgravity environment, reducing symptoms. Moreover, development of the framework to investigate other multisensory conflicts or even

conflicts within modalities (Bles, 1998; Keshavarz, Hecht, & Zschutschke, 2011) may further enhance our understanding of wider motion sickness.

Limitations and Future Directions

Firstly, one limitation to this work relates to the time-course of the effects described. As previously noted, the after-effects of VR are not well described in the literature. As such, we do not fully understand the true extent of VR after-effects, nor is there extensive evidence for their development. In Chapters 4 and 5, I described changes in both high and low-level vestibular processing. These effects arose from exposure to approximately one minute of VR exposure, however typically exposure to VR is over much longer periods. Moreover, cybersickness tends to develop with increasing exposure (Liu, 2014; Moss et al., 2011; Stanney et al., 1999). Accordingly, studies of VR after-effects and cybersickness immerse participants for upwards of 15 minutes (Di Girolamo et al., 2001; Harm et al., 2008; Liu, 2014). Here, it is not possible to know whether the changes in vestibular processing will remain the same, improve, or worsen with increasing exposure to VR. In addition, understanding whether the effects persist over time as the participant is exposed to the real world on exiting VR is vital. In the work presented here, I measured vestibular processing after one minute of adaptation to VR stimuli, however this was not measured in the absence of visual stimuli following exposure. Moreover, I did not find changes in VEMPs following exposure to a VR driving simulator, suggesting that differences in timing and/or the VR scenarios may have different impacts on VR after-effects. Thus, future work is necessary, both extending the duration of exposure to VR and measuring vestibular functioning over time following exposure.

Secondly, it is important to consider that the visuo-vestibular multisensory integration framework presented here is based on the notion that visual and vestibular cues for self-motion are in conflict during VR exposure. While many commercial VR applications do indeed make use of vection-inducing visual stimuli to enhance the immersive experience of VR, other categories of application may not. As such, VR scenarios in which the user does not move around the virtual environment, or scenarios in which users can also physically navigate through the real world may differ from cases where visual cues signal that the user is moving when they are in fact stationary. The visuo-vestibular framework might predict that these cases would have lower levels of cybersickness than conflicting VR applications, given the reduction in sensory conflict. The fact that locomotion may reduce cybersickness is evidence for this, however findings are mixed (Chance et al., 1998; Llorach et al., 2014; Peck et al., 2011). Moreover, while I focused on VR presented on head-mounted displays, there are many other VR formats, such as CAVEs or large projectors which can induce cybersickness (Kennedy, Drexler, & Kennedy, 2010; Whitton et al., 2005). While it is assumed that the mechanisms are similar across VR types, a direct test of the visuo-vestibular framework for these alternative setups will be necessary to confirm this.

Finally, a key limitation to the current work is the predominant focus on visuo-vestibular integration. While these two sensory modalities are key for the perception of self-motion (DeAngelis & Angelaki, 2012; Greenlee et al., 2016), it is important to consider that a host of other signals, such as proprioceptive, somatosensory and auditory cues, are also implicated. Moreover, modern VR applications can also incorporate additional sensory cues to render the virtual environment more immersive. For instance, haptic stimulators are proving particularly useful in VR applications designed to enhance training for surgeons (Kim, Kim, & Kim, 2017; Zaragoza-

Siqueiros, Medellin-Castillo, de la Garza-Camargo, Lim, & Ritchie, 2019), and VR applications which incorporate additional multisensory cues are rated as more immersive and can improve performance on the VR task (Cooper et al., 2018). Moreover, while vection is frequently induced by visual cues, it is also possible to induce the illusion by auditory cues alone (Riecke, Feuereissen, Rieser, & McNamara, 2015; Väljamäe, 2009). As such, for the multisensory integration framework to truly capture cybersickness, VR adaptation, and after-effects, future work must also consider the impact of these additional multisensory cues.

In addition to the limitations discussed above, it is also important to discuss the potential limitations of the sample sizes employed in the thesis. Sample sizes across the empirical chapters varied from 8 (Chapter 8), 15 (Chapter 7), to 24 participants (Chapters 4-6). These sample sizes may at first glance be considered low, potentially limiting the generalisability and replicability of the findings. However, several factors were taken into consideration when deciding on the sample sizes of the present studies, which aimed to ensure the reliability and replicability of the results.

All sample sizes were chosen a priori on the basis of previous similar results. For instance, sample sizes investigating the impact of optic flow on vestibular processing have ranged from four (Edwards et al., 2010) to 14 participants (Holten & MacNeilage, 2018), while studies investigating VEMPs processing and motion sickness range from 24-30 (Fowler et al., 2014; Tal et al., 2013). Accordingly, the sample sizes of in Chapter 4 and 5 are comparable to previous studies, and are likely to have sufficient power to detect significant effects. Moreover, the relatively large effect sizes in both of these studies and the fact that they accord well with previous studies investigating the effect of VR on vestibular processing (Di Girolamo et al., 2001) suggest that these results are likely to be reliable.

Studies investigating vestibular stimulation for cybersickness reduction tend to have higher sample sizes, from 19 (Reed-Jones et al., 2007) to 40 (Weech, Wall, & Barnett-Cowan, 2020). Thus, it is possible that the numbers of participants were low in Chapters 6 and 7. However, a power analysis for Chapter 6 suggested a sample size of 18 would be sufficient to detect a significant effect, and it is possible that the null findings are due to other factors, such as habituation to the tilted body posture. A within-subjects design was used in Chapter 7, increasing statistical power. However, carry-over effects between sessions may have masked potential effects of GVS on cybersickness. Accordingly, the null findings in these two chapters are likely to require further exploration, potentially with increased sample sizes.

In Chapter 8, a smaller sample size of eight participants was used, largely due to technical constraints regarding the combination of artificial and natural vestibular stimulation. While this is a small sample size, it is important to note that this study employed a large number of trial repetitions with psychophysical methods, a design whereby repeated sessions were conducted within-subjects, and intra- rather than inter-subject variability was of interest in this experiment, potentially mitigating negative effects of a smaller sample size (Smith & Little, 2018). Furthermore, linear mixed models which incorporated the participant as a random factor were used to analyse data, accounting for variability arising from individual subjects. Thus, while further replications with larger samples are necessary to confirm the generalisability of these findings, the low sample size is not necessarily a prohibitive factor to the reliability of the results (Smith & Little, 2018).

Overall Evaluation

The central argument of my thesis is that the brain dynamically re-weights vestibular cues for self-motion during and after exposure to visuo-vestibular conflicts in VR. This dynamic re-weighting leads to reduced cybersickness, but may subsequently contribute to after-effects of VR exposure. To investigate this proposal, I explored vestibular processing during exposure to optic flow in VR (Chapters 4 and 5), and investigated vestibular physiologically-driven interventions for cybersickness (Chapters 6 and 7). Finally, I quantified the illusory self-motion induced by artificial vestibular stimulation (Chapter 8), which could be used to refine vestibular-driven cybersickness interventions. Taken together, my findings largely support the notion of a visuo-vestibular multisensory integration framework for cybersickness, however further research is necessary to investigate remaining gaps.

In Chapter 4, I found that sensitivity to vestibular cues was substantially decreased following exposure to optic flow, however this was only the case when optic flow signalled self-motion on the same axis as the vestibular cues. In Chapter 5, I extended the findings of Chapter 4 by highlighting how vestibular physiological processing was also significantly altered by exposure to visuo-vestibular conflicts in VR. Specifically, VEMPs asymmetries were significantly increased following exposure to linear optic flow. Taken together, these findings provide evidence for a dynamic re-weighting of vestibular cues following exposure to optic flow in VR, however to fully explore this idea these results should be replicated following exposure to complex optic flows (Smith, Wall, Williams, & Singh, 2006; Uesaki & Ashida, 2015), real VR scenarios (Reed-Jones et al., 2007; Cevette et al., 2012), and longer time-courses of exposure (Liu, 2014; Moss et al., 2011). Interestingly, the findings also suggest that the dynamic re-weighting of vestibular cues is dependent on the

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congruency between visual and vestibular cues for self-motion, with down-weighting specifically for vestibular cues signalling self-motion on the same axis as visual cues. However, this specificity was only tested for vestibular sensitivity, and accordingly future research is necessary to investigate whether this effect is also apparent in vestibular physiological measures. For instance, while we found a change in VEMPs asymmetry during exposure to linear (i.e., congruent) optic flow, we might predict that VEMPs would be unaffected during exposure to an incongruent optic flow, such as roll. Ultimately, while sensitivity to artificial vestibular stimulation and VEMPs are proxies for assessing the weight of vestibular cues, a direct exploration of vestibular weighting will be necessary to confirm whether vestibular re-weighting is indeed the mechanism for these VR-induced changes. Thus, one might envisage a study whereby vestibular weights for heading direction (DeAngelis & Angelaki, 2012; Fetsch, Turner, DeAngelis, & Angelaki, 2009; Gu, Angelaki, & DeAngelis, 2008) or verticality perception (Alberts, de Brouwer, Selen, & Medendorp, 2016; Alberts, Selen, et al., 2016) are measured before and after exposure to VR. However, such a study would be technically challenging, due to the need for complex vestibular stimulation equipment, and the large number of trials and conditions necessary to generate the predicted vestibular weightings (Fetsch, DeAngelis, & Angelaki, 2010; Rohde, van Dam, & Ernst, 2015) even without exposure to VR. This therefore remains an open question.

As well as investigating vestibular re-weighting following VR exposure, I also investigated interventions for cybersickness based on predictions from the visuo-vestibular framework. Specifically, the framework suggests that either inducing vestibular down-weighting before VR exposure or preventing visuo-vestibular conflicts during exposure should result in reduced cybersickness. In Chapter 6, I investigated the former by tilting participants away from the gravitational vertical. In

this body posture, the reliability of otolith cues for gravity is reduced, potentially resulting in a lower relative weighting of vestibular cues with respect to other cues, including vision (Alberts, de Brouwer, Selen, & Medendorp, 2016; Alberts, Selen, et al., 2016). In Chapter 7, artificial vestibular stimulation was used as a mechanism for prevention of visuo-vestibular conflicts during a VR driving simulator. Specifically, GVS was applied during turns of the driving simulator, potentially resulting in congruent vestibular and visual cues for self-motion. While numerical differences in cybersickness scores were found in both studies, no significant effects were apparent. Accordingly, it is not possible to argue that these findings fully support the visuo-vestibular framework. However, as previously discussed, further refinement of both interventions may be necessary. Moreover, a number of previous studies support both mechanisms for cybersickness reduction. For example, both Bone Conducted Vibration (BCV) and noisy GVS have been shown to reduce cybersickness in VR (Weech, Moon, & Troje, 2018; Weech, Wall, & Barnett-Cowan, 2020), potentially due to a reduction in vestibular reliability. In addition, GVS congruent with visual cues has also been shown to reduce scores of simulator sickness (Reed-Jones et al., 2007; Cevette et al., 2012). Thus, these findings support the predictions of the visuo-vestibular integration framework, even though the current interventions prove inconclusive.

Given the limitations of GVS for cybersickness reduction, in Chapter 8 I quantified the illusory motion induced by GVS. We found that GVS elicited a roll sensation towards the cathode of approximately 2 deg/s and 6 deg/s velocity at 1mA and 2.5mA of square-wave GVS. These results were found when participants experienced GVS while supine, to reduce confounds of proprioceptive and somatosensory cues for gravity (Vimal, DiZio, & Lackner, 2017). In addition, they

were elicited from binaural-bipolar GVS, which elicits specific sensations of roll, while other configurations of GVS can also elicit sensations of pitch and yaw rotation (Aoyama, Iizuka, Ando, & Maeda, 2015). Thus, this work should be extended to other axes of rotation and body postures if GVS is to be used as a mechanism for cybersickness reduction. However, it is important to note that the vestibular system provides dynamic, complex sensations of self-motion. Given the sophistication of the vestibular system, it may not be possible to fully replicate in VR the sensations of motion that would be present in everyday motion. Accordingly, more research is necessary to find the limits at which GVS for cybersickness reduction is possible.

Taken together, my findings provide evidence for re-weighting of vestibular cues following exposure to visuo-vestibular conflicts in VR. Findings regarding interventions for cybersickness are perhaps less conclusive and further research is necessary, however previous literature supports interventions based on similar mechanisms (Cevette et al., 2012; Reed-Jones, Reed-Jones, Trick, & Vallis, 2007; Weech, Moon, & Troje, 2018; Weech, Wall, & Barnett-Cowan, 2020), further substantiating the visuo-vestibular framework for cybersickness. To fully explore the visuo-vestibular framework, several open questions remain. Firstly, findings from the present thesis should be extended to more complex VR and/or optic flow displays over longer time-courses to explore the limits of visuo-vestibular reweighting. For instance, while I found that re-weighting is specific to the axis of motion presented, it is unclear whether similar findings are extended to more complex optic flows or real VR scenarios. In addition, the spatio-temporal limits of the visuo-vestibular conflict which induces vestibular re-weighting should be considered. While I investigated conflicts based on optic flows signalling self-motion along a particular spatial dimension, it is also possible that temporal conflicts (for example, lags of the VR scene as a user

physically moves through an environment) could also trigger vestibular re-weighting. In addition, future research must also consider the role of individual differences in vestibular weighting on cybersickness severity. Specifically, the visuo-vestibular framework predicts that individuals who are more reliant on vestibular cues may be more susceptible to symptoms of cybersickness (Keshavarz, Speck, Haycock, & Berti, 2017). Finally, the present thesis and framework is outlined only in terms of visuo-vestibular conflicts, but it is possible that additional modalities may contribute to cybersickness. For instance, it could be that audio-vestibular or visuo-proprioceptive conflicts in VR could also contribute to cybersickness, and re-weighting of cues from other sensory modalities could result in VR adaption. Exploration of these remaining gaps should therefore provide a more comprehensive analysis of the visuo-vestibular multisensory integration framework for cybersickness.

Conclusion

The multisensory integration framework I have outlined in my thesis represents a clear advance in cybersickness research. Specifically, it provides clear, testable hypotheses to investigate mechanisms of cybersickness development, adaptation to VR, after-effects of VR, and methods to prevent symptoms. Moreover, the studies conducted in my thesis provide clear evidence of vestibular processing modulation by VR, contributing to our understanding of VR after-effects and general multisensory integration mechanisms. Although the techniques to prevent cybersickness did not yield significant reductions in sickness, it is likely that refinement of these techniques will lead to new methods to reduce sickness and improve the user experience in VR. The prospect of doing this in the absence of technological solutions by modifying posture is the first cybersickness prevention technique that is likely to be truly

accessible to all. Finally, I have also provided empirical evidence for a natural equivalent motion to GVS, answering theoretical questions regarding the nature of stimulation. These findings can further optimise GVS for VR, as well as answering wider theoretical questions regarding the technique.

Limitations of my thesis work include a focus on visuo-vestibular integration, while cybersickness may also entail other multisensory cues. Moreover, a clear understanding of individual factors and the time-course of cybersickness and VR after-effects is not provided by the work presented here. Finally, refinements of the cybersickness prevention methods are necessary to investigate their efficacy further. Accordingly, my thesis provides a range of future avenues for research. Specifically, an investigation of other multisensory interactions in VR and their contributions to cybersickness is warranted, as is an investigation of individual differences in multisensory re-weighting in cybersickness development. Moreover, exploration of the time-course of VR after-effects, as well as the specificity of the visuo-vestibular conflict will further enhance our understanding of cybersickness and VR after-effects.

The developments in VR technology have been vast in recent decades. VR applications provide exciting potential for education, training, rehabilitation, and research, as well as more general recreational and entertainment purposes. Given these beneficial applications, and the clear economic impact of VR, it is vital that the VR user experience is comfortable for all. Despite technological improvements, cybersickness remains a significant problem. Moreover, we are only beginning to understand the true extent of VR-induced after-effects. Accordingly, my thesis provides a framework for understanding and exploring cybersickness and VR after-effects, as well as clear evidence for alterations in vestibular processing. These

findings therefore add to our understanding of the sensory implications of VR and will

prove useful for researchers and VR developers alike.

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EDINBURGH HANDEDNESS INVENTORY

NAME	
AGE	
SEX	
DATE	

Please indicate your preference in the use of hands in the following activities.

Task / Object	Strong left preference	Left preference	No preference	Right preference	Strong right preference
1. Writing					
2. Drawing					
3. Throwing					
4. Scissors					
5. Toothbrush					
6. Knife (without fork)					
7. Spoon					
8. Broom (upper hand)					
9. Striking a Match (match)					
10. Opening a Box (lid)					
11. Which foot do you kick with?					
12. Which eye do you use when using only one eye?					

Appendix 2: Motion Sickness Susceptibility Questionnaire

This questionnaire is designed to find out how susceptible to motion sickness you are, and what sorts of motion are most effective in causing that sickness. Sickness here means feeling queasy or nauseated or actually vomiting.

Your CHILDHOOD Experience Only (before 12 years of age), for each of the following types of transport or entertainment please indicate

As a CHILD (before age 12), how often you Felt Sick or Nauseated (tick boxes):

	Not Applicable/ Never Travelled	Never felt sick	Rarely felt sick	Sometimes felt sick	Frequently felt sick
Cars					
Buses or Coaches					
Trains					
Aircraft					
Small boats					
Ships, e.g. channel ferries					
Swings in playgrounds					
Roundabouts in playgrounds					
Big dippers, funfair rides					
	t	0	1	2	3

Your Experience over the LAST 10 YEARS (approximately), for each of the following types of transport or entertainment please indicate:

Over the LAST 10 YEARS, how often you Felt Sick or Nauseated (tick boxes):

	Not Applicable/ Never Travelled	Never felt sick	Rarely felt sick	Sometimes felt sick	Frequently felt sick
Cars					
Buses or Coaches					
Trains					
Aircraft					
Small boats					
Ships, e.g. channel ferries					
Swings in playgrounds					
Roundabouts in playgrounds					
Big dippers, funfair rides					
	t	0	1	2	3

Appendix 3: Simulator Sickness Questionnaire

Check the items that apply to you at this time:

	None	Slight	Moderate	Severe
General discomfort				
Fatigue				
Headache				
Eye strain				
Difficulty focusing				
Increased salivation				
Sweating				
Nausea				
Difficulty concentrating				
"Fullness of the head"				
Blurred vision				
Dizzy (eyes open)				
Dizzy (eyes closed)				
Vertigo				
Stomach awareness*				
Burping				
Other (describe):				

Appendix 4: Supplementary Dot Plots

Chapter 4: Supplementary Dot Plots
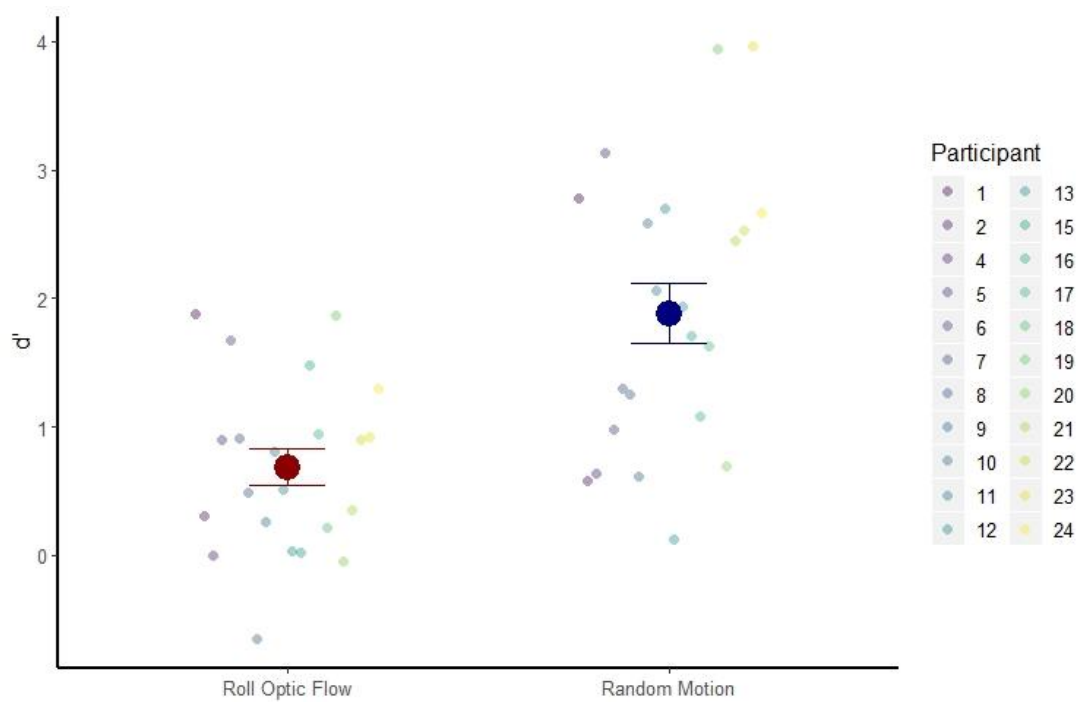
Figure A1. Chapter 4, Experiment 1: Sensivity (d') Results. Vestibular sensitivity was significantly reduced following exposure to congruent optic flow in VR. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
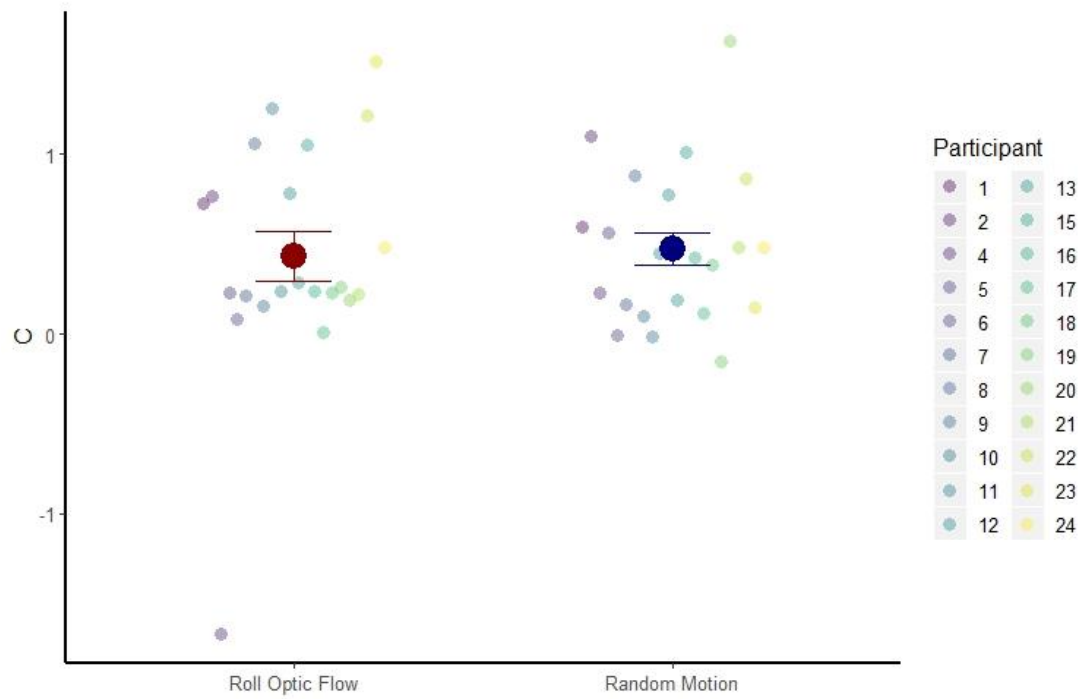
Figure A2. Chapter 4, Experiment 1: Response Bias (C) Results. Response bias was not significantly different following exposure to congruent optic flow or random motion in VR. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
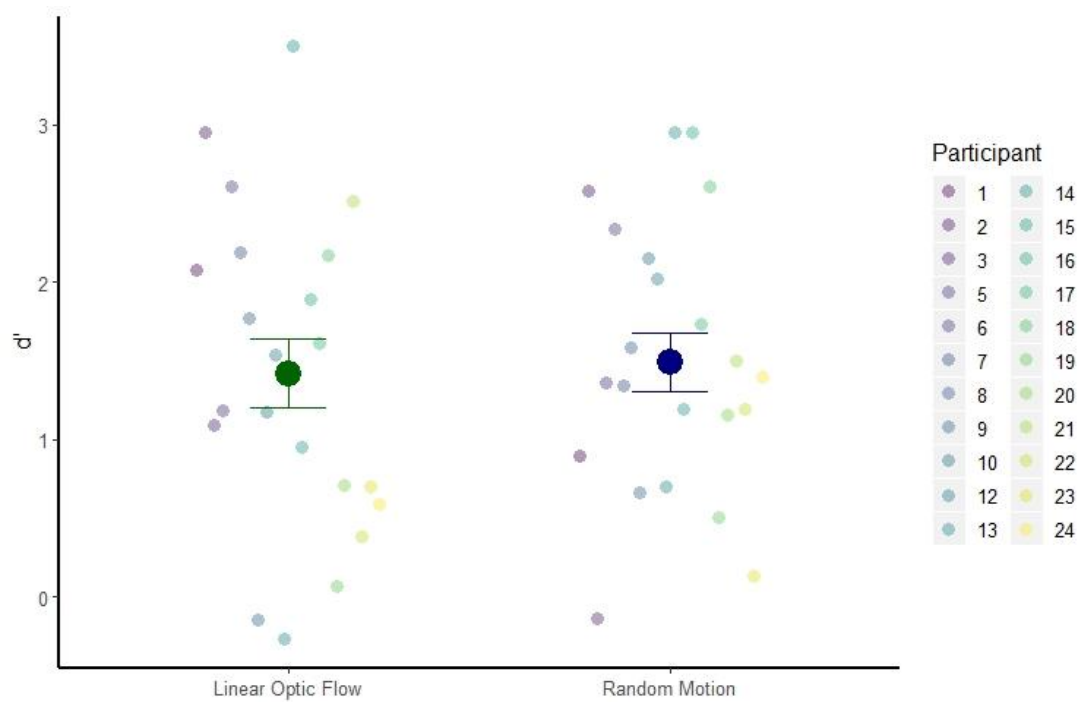
Figure A3. Chapter 4, Experiment 2: Sensivity (d') Results. Vestibular sensitivity was not significantly different following exposure to incongruent optic flow versus random motion in VR. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
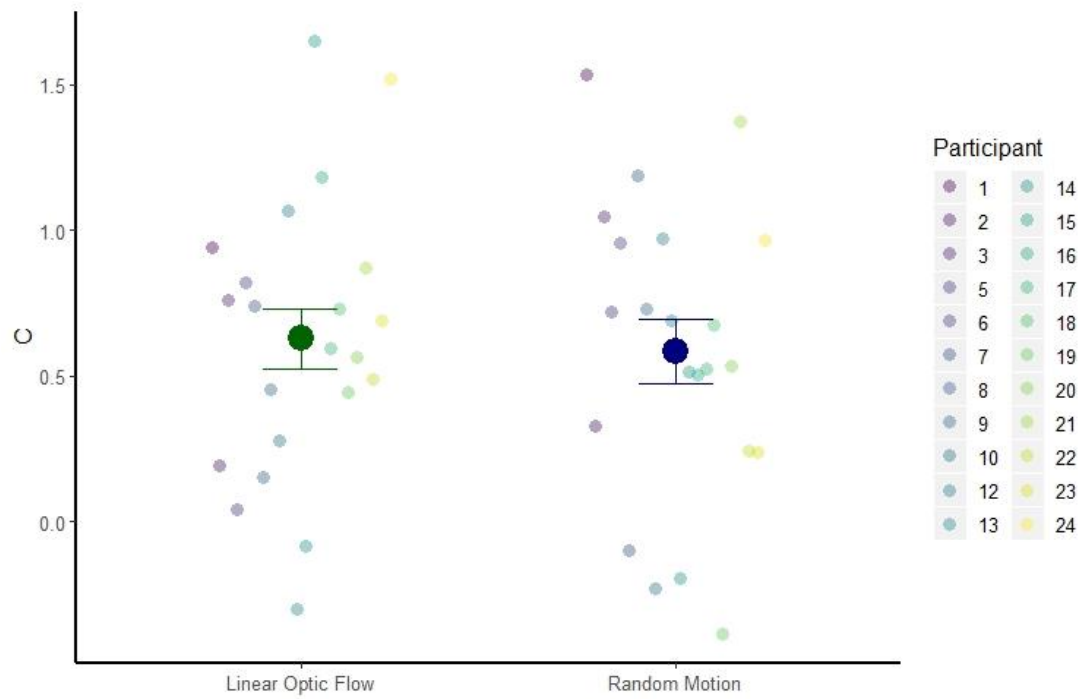
Figure A4. Chapter 4, Experiment 2: Response Bias (C) Results. Response bias was not significantly different following exposure to incongruent optic flow or random motion in VR. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
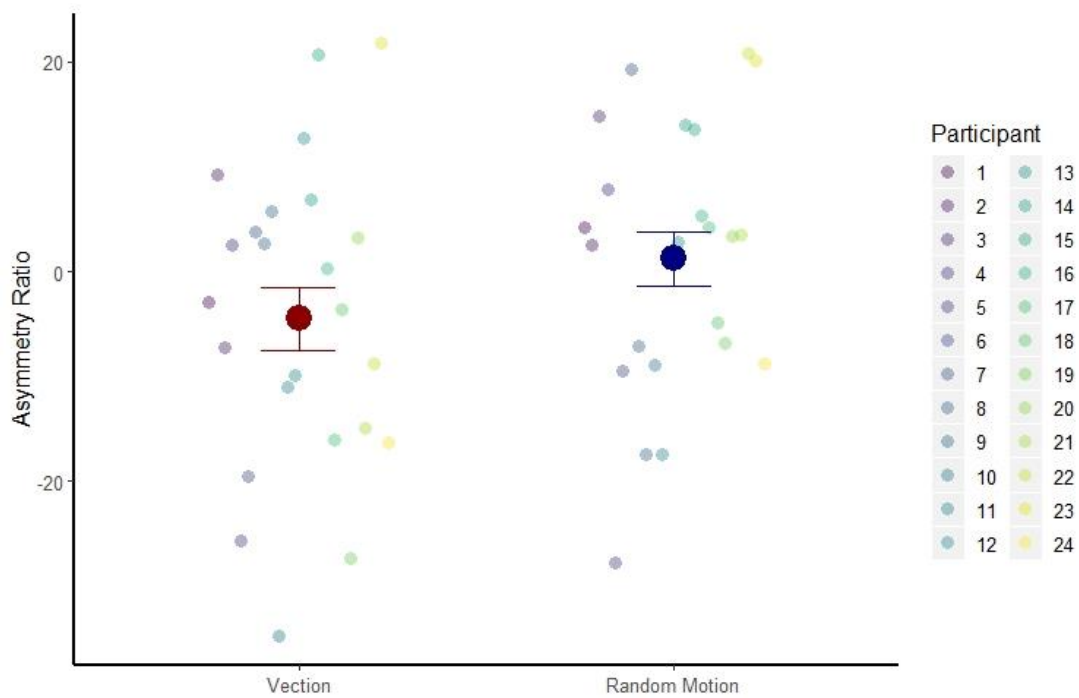
Figure A5. Chapter 5, VEMPs Asymmetry Ratios. VEMPs asymmetry significantly increased following exposure to vection in Virtual Reality relative to random motion, with larger amplitudes on the left muscle side. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
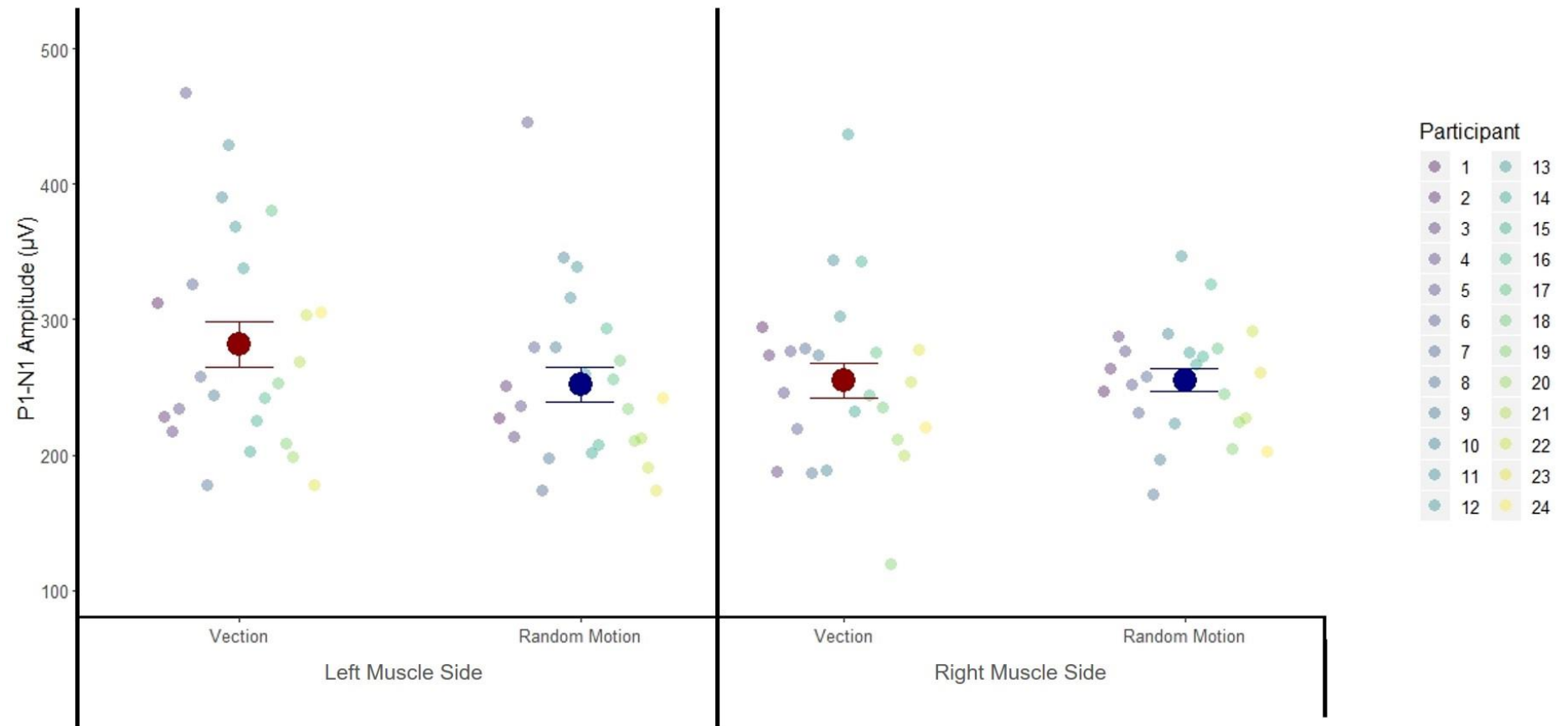
Figure A6. Chapter 5, VEMPs Amplitudes. P1-N1 peak-to-peak amplitude significantly increased on the left muscle side following exposure to vection in Virtual Reality relative to random motion. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
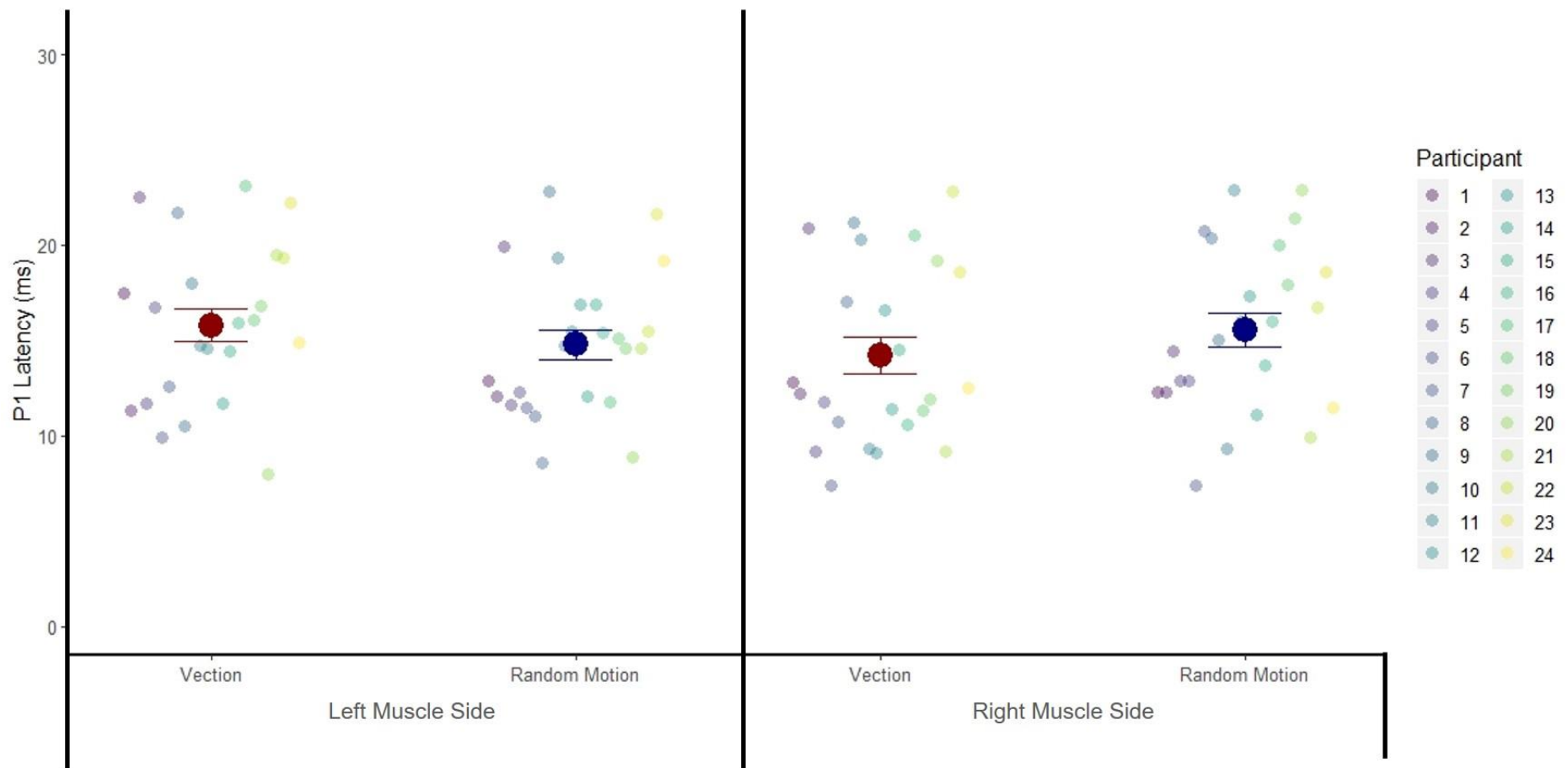
Figure A7. Chapter 5, VEMPs P1 Latency. No significant changes were found in P1 latencies. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.

Figure A8. Chapter 5, VEMPs N1 Latency. No significant changes were found in N1 latencies. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
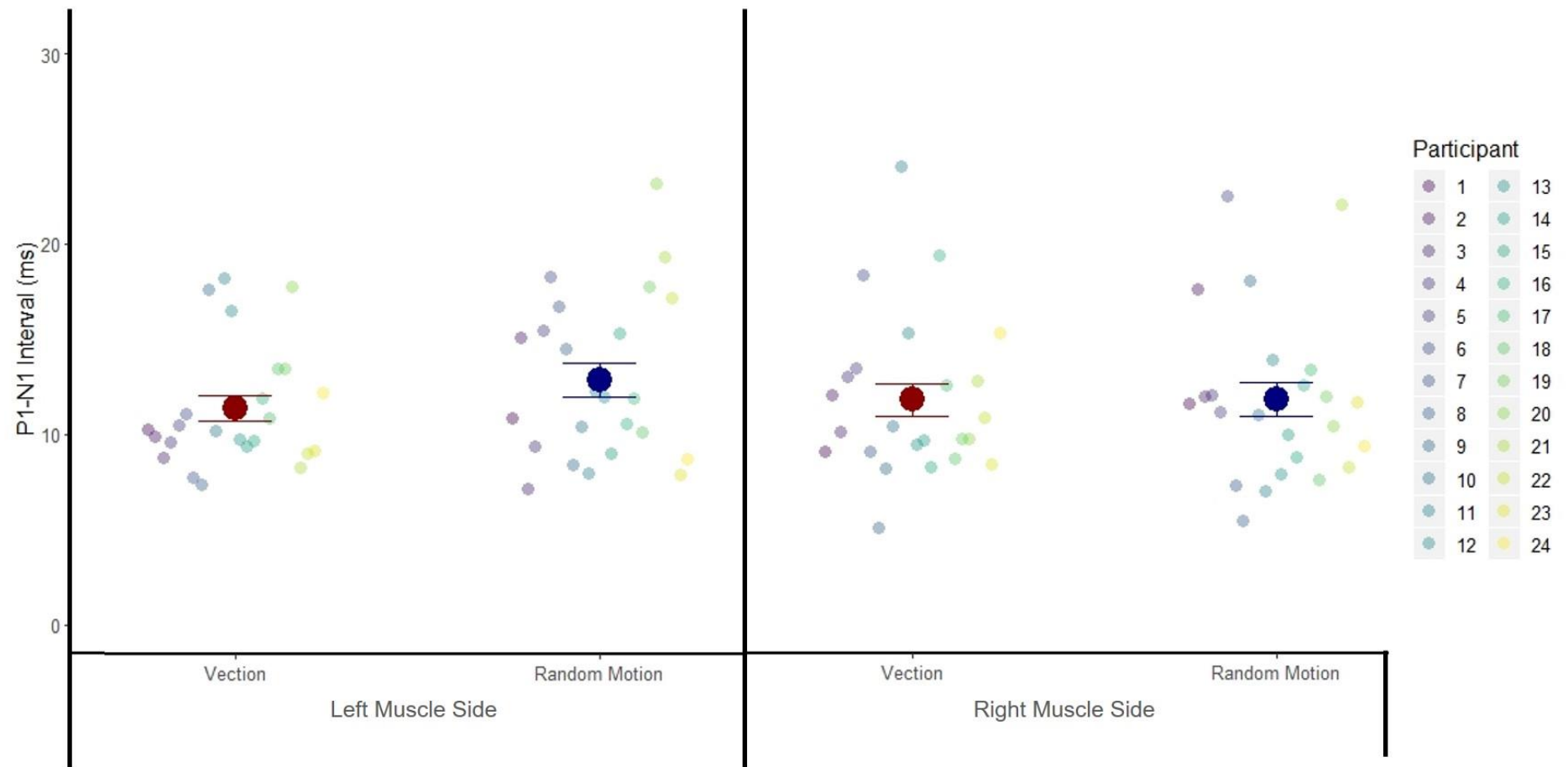
Figure A9. Chapter 5, VEMPs P1-N1 Interval. No significant changes were found in P1-N1 intervals. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
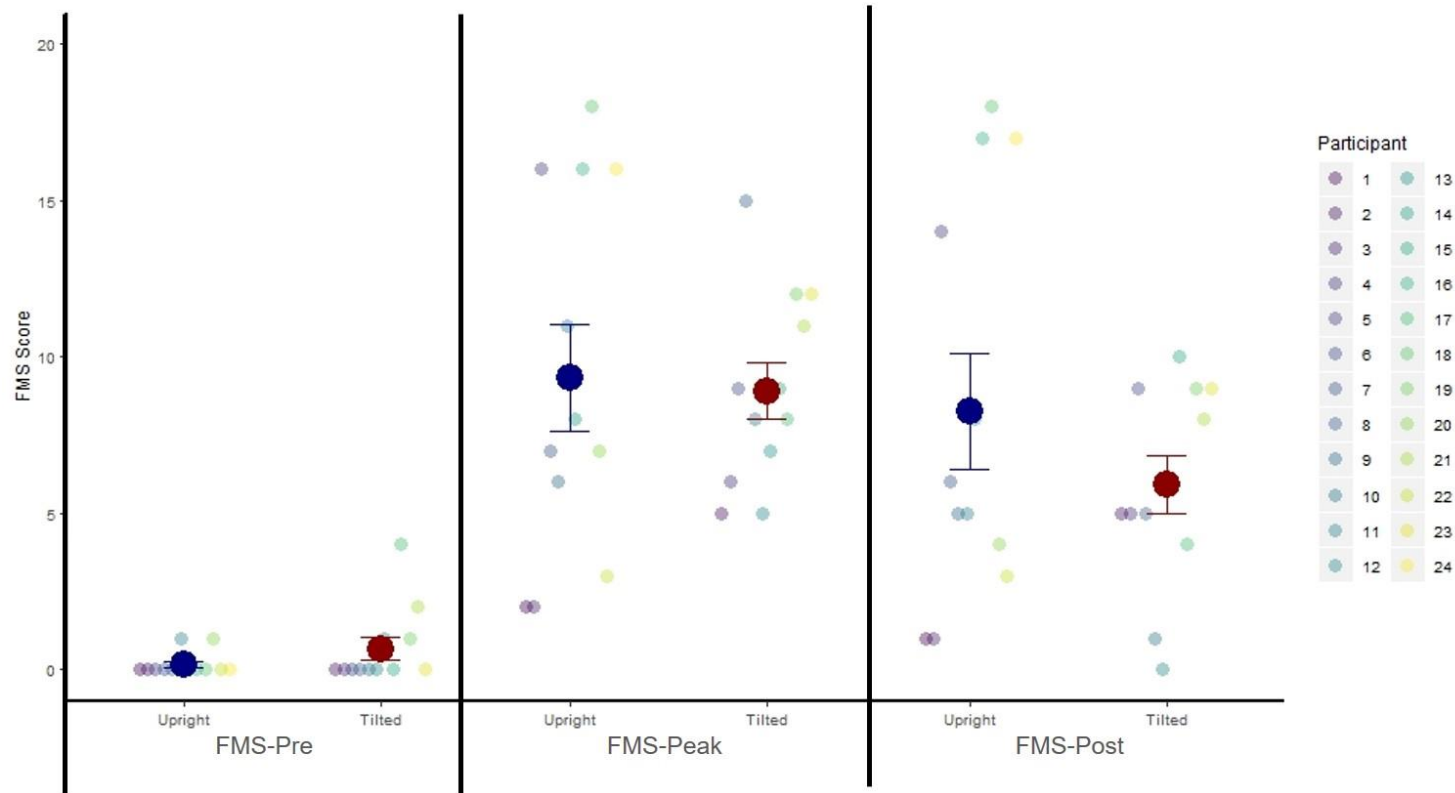
Figure A10. Chapter 6, Fast Motion Sickness Results. Peak FMS scores were significantly higher than scores Pre- and Post-VR, while Post- FMS scores were also significantly higher than Pre-VR scores. No significant effect of body orientation was found. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
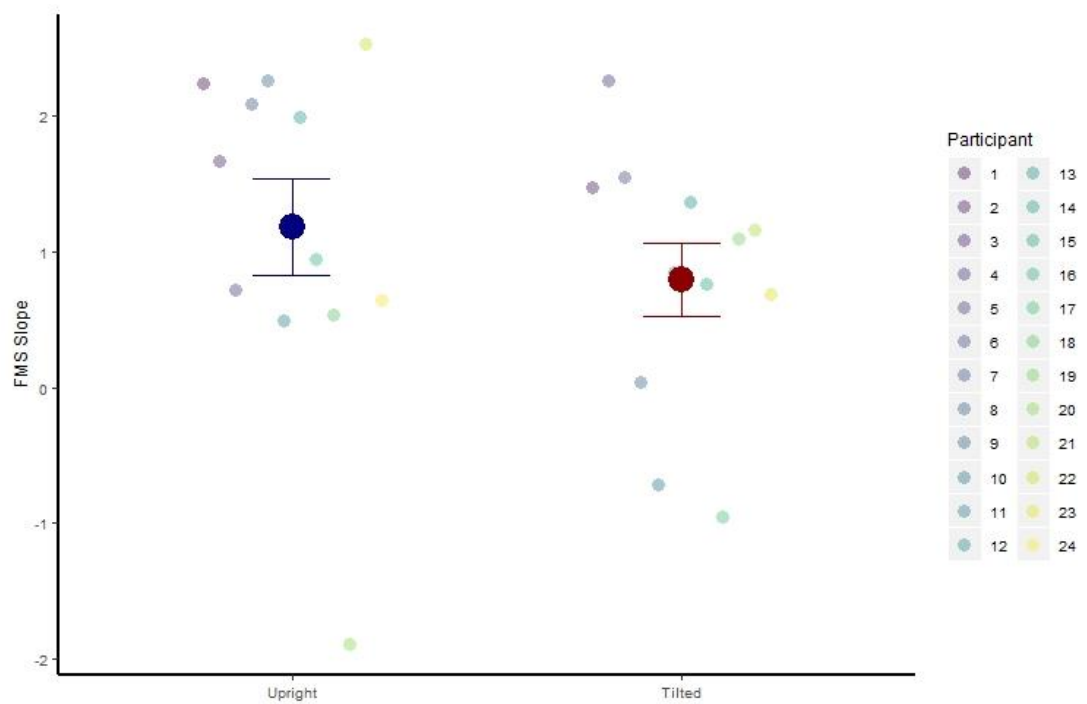
Figure A11. Chapter 6, Fast Motion Sickness Slopes. No significant changes were found on FMS slopes between body postures. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
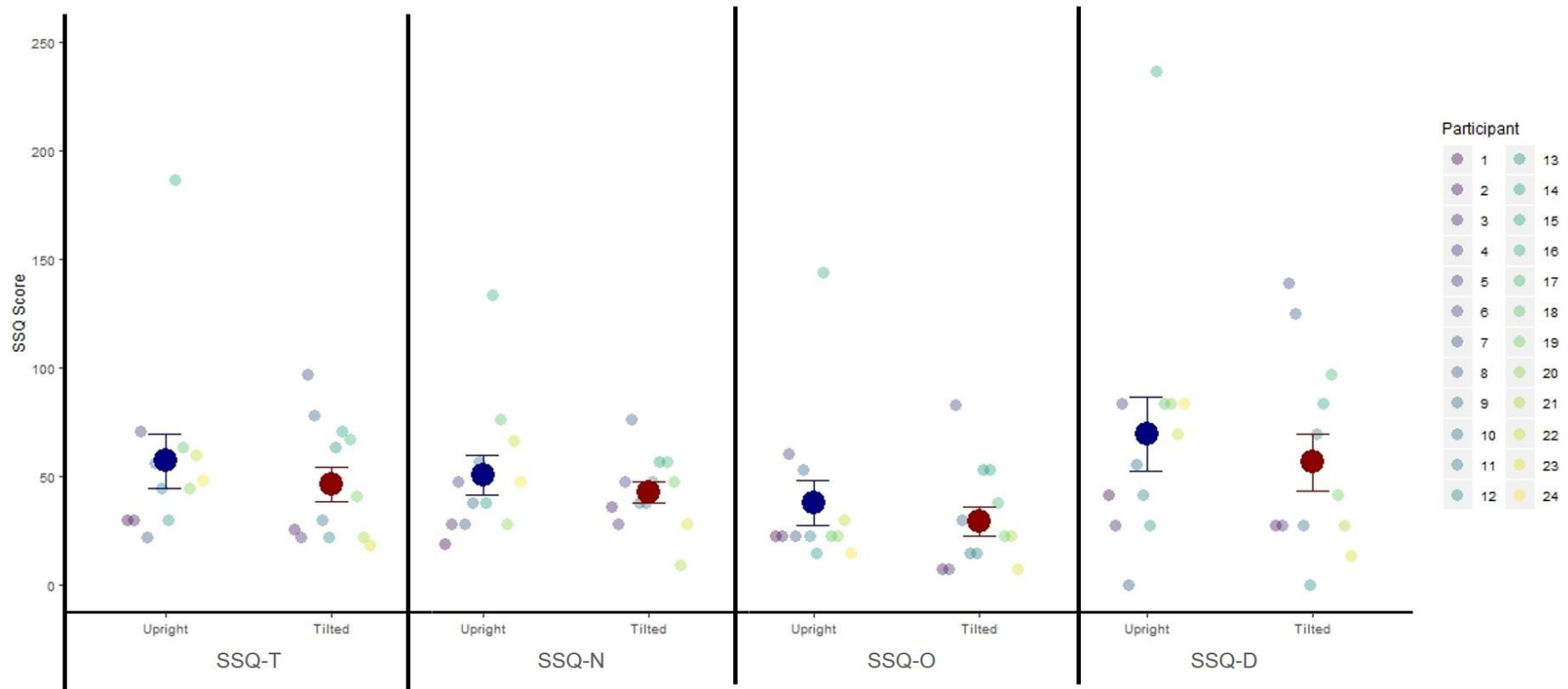
Figure A12. Chapter 6, Simulator Sickness Questionnaire Results. No significant changes were found on SSQ Total or Subscale scores between body postures. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
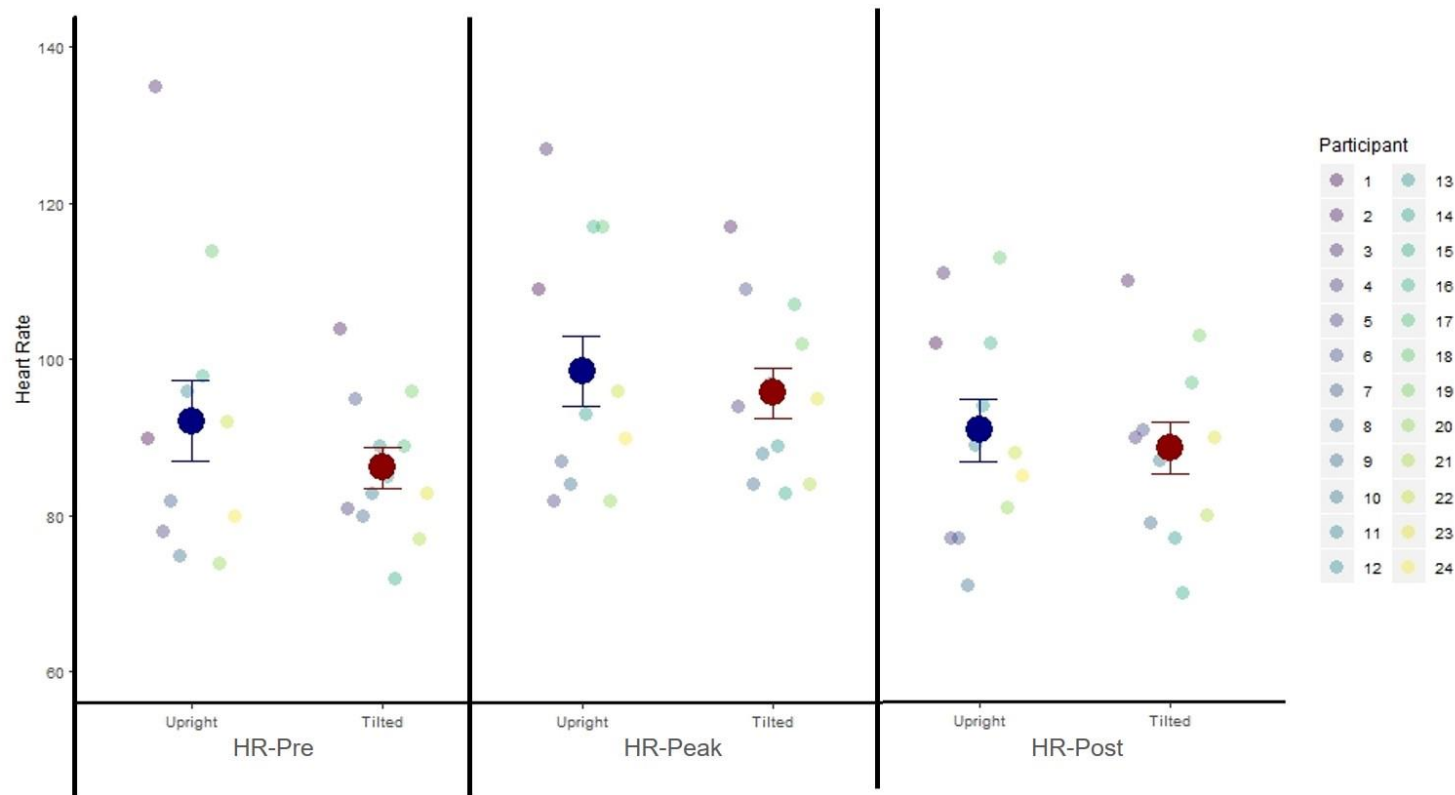
Figure A13. Chapter 6, Heart Rate Results. Peak heart rate was significantly higher than Pre- and Post-VR. No significant effect of body orientation was found. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
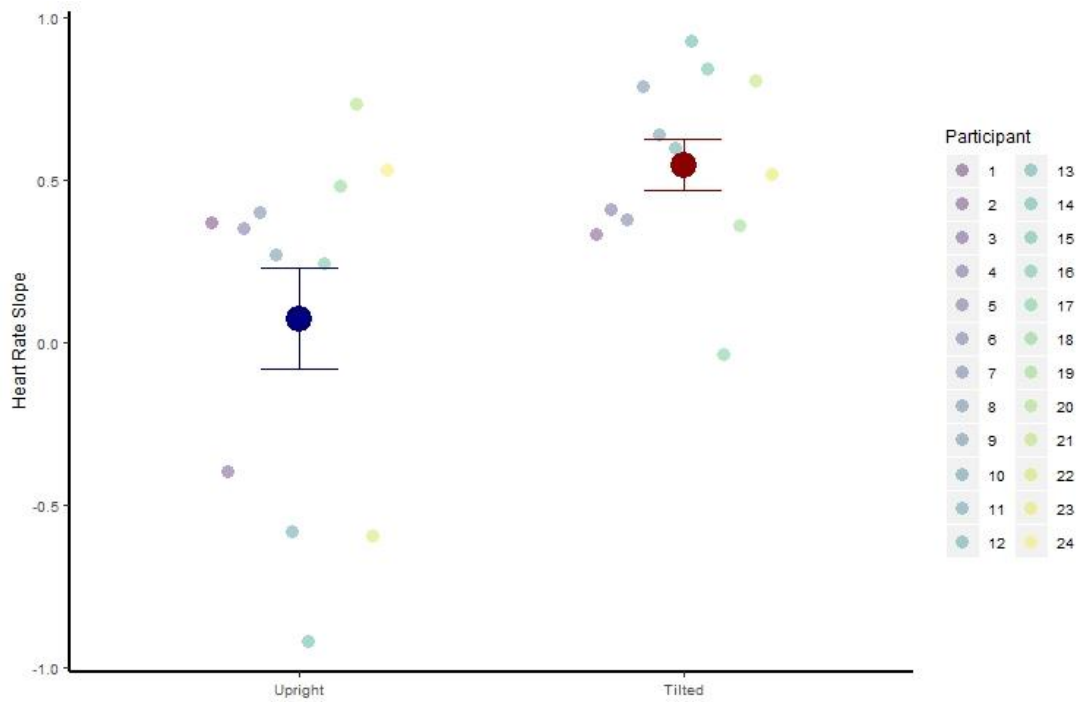
Figure A14. Chapter 6, Heart Rate Slopes. Slopes for the Tilted body orientation were signfiicantly higher than the Upright body orientation. This difference may be driven by lower heart rates for participants in the Tilted condition in the early During-VR time points. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
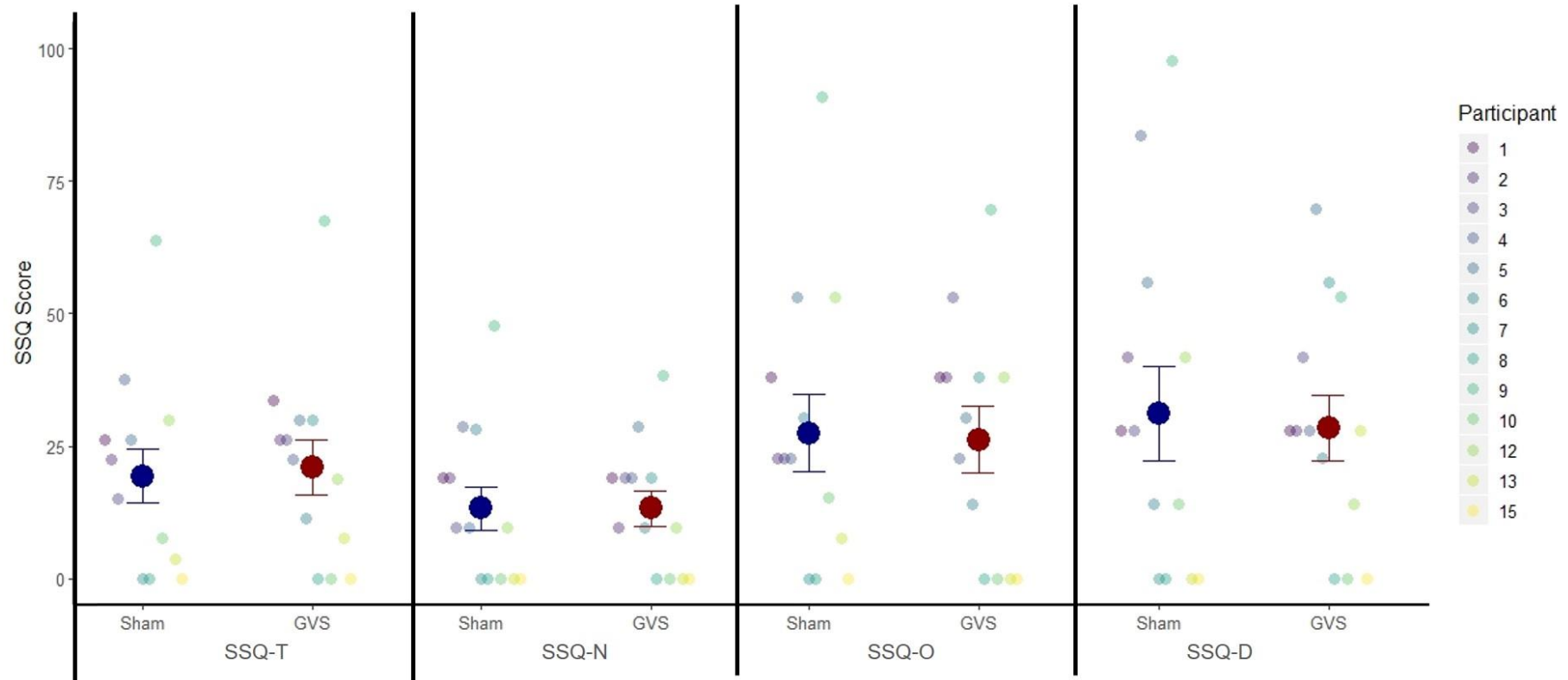
Figure A15. Chapter 7, Simulator Sickness Questionnaire Results. No significant changes were found on SSQ Total or Subscale scores between body GVS and Sham conditions. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
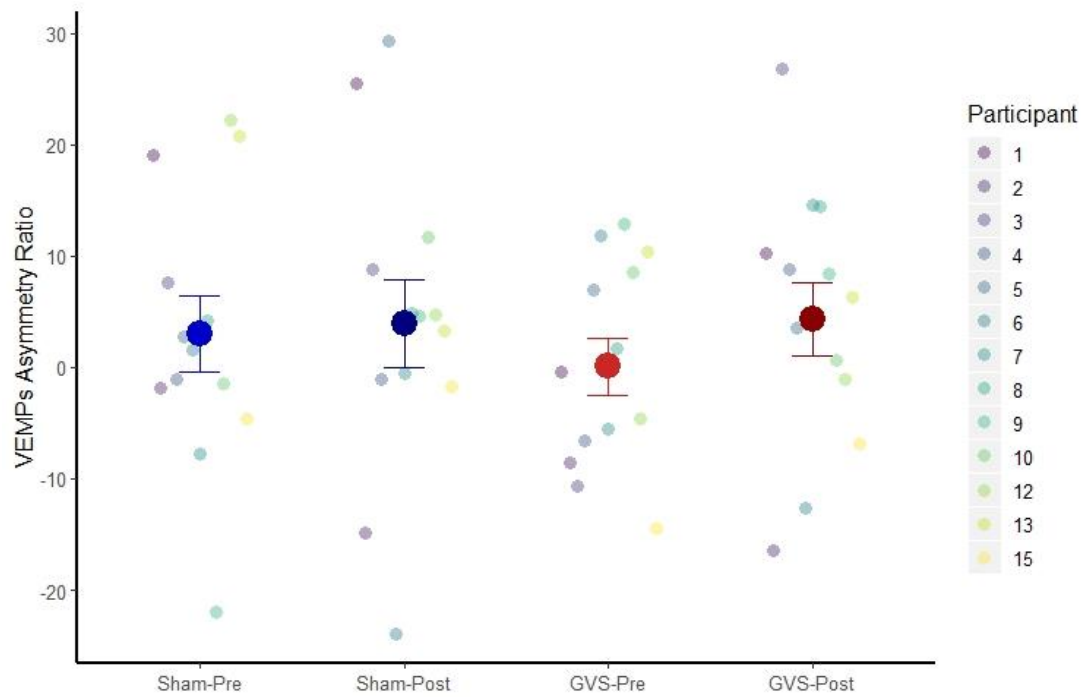
Figure A16. Chapter 7, VEMPs Asymmetry Ratios. No significant changes in VEMPs asymmetry ratios were found following exposure to VR with GVS or Sham stimulation. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
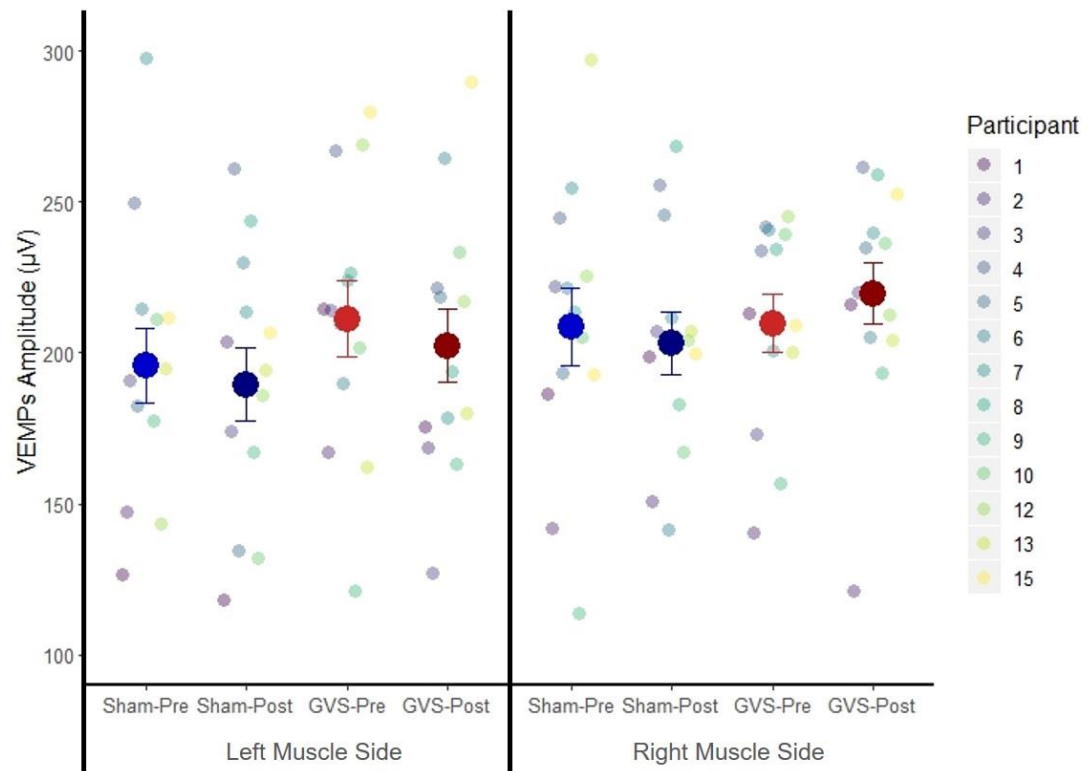
Figure A17. Chapter 7, VEMPs Amplitudes. No significant changes in VEMPs amplitudes were found following exposure to VR with GVS or Sham stimulation. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
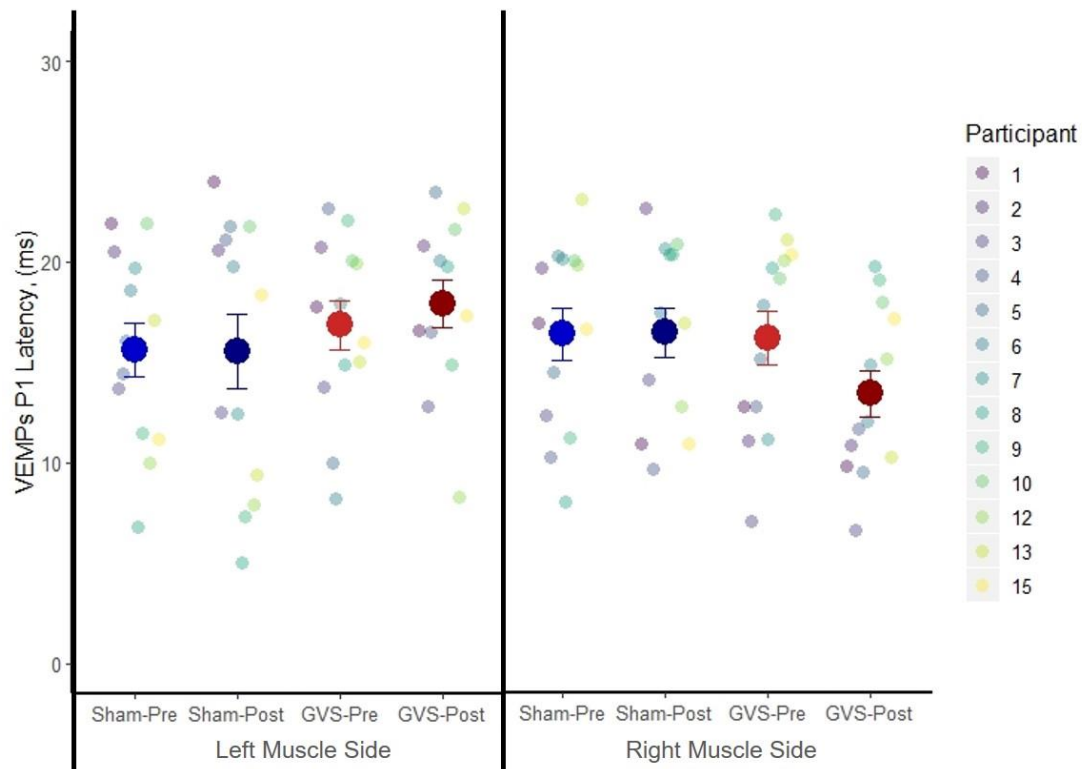
Figure A18. Chapter 7, P1 Latency. No significant changes in VEMPs P1 Latencies were found following exposure to VR with GVS or Sham stimulation. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
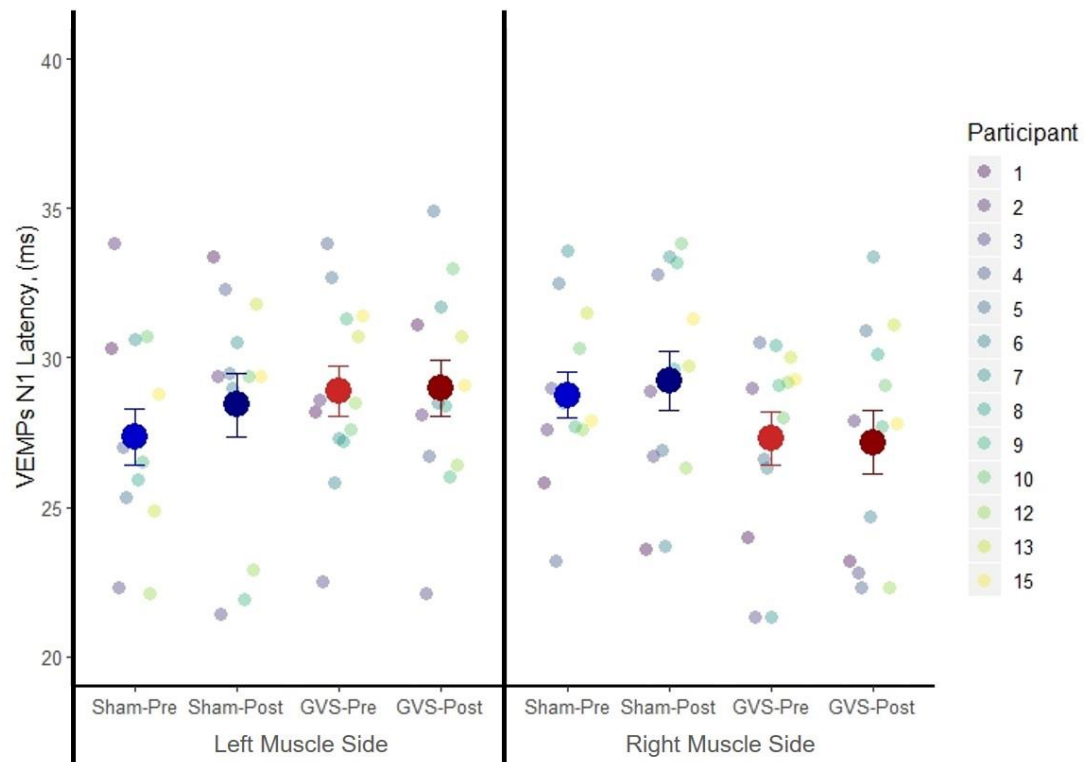
Figure A19. Chapter 7, VEMPs N1 Latency. No significant changes in VEMPs N1 Latencies were found following exposure to VR with GVS or Sham stimulation. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
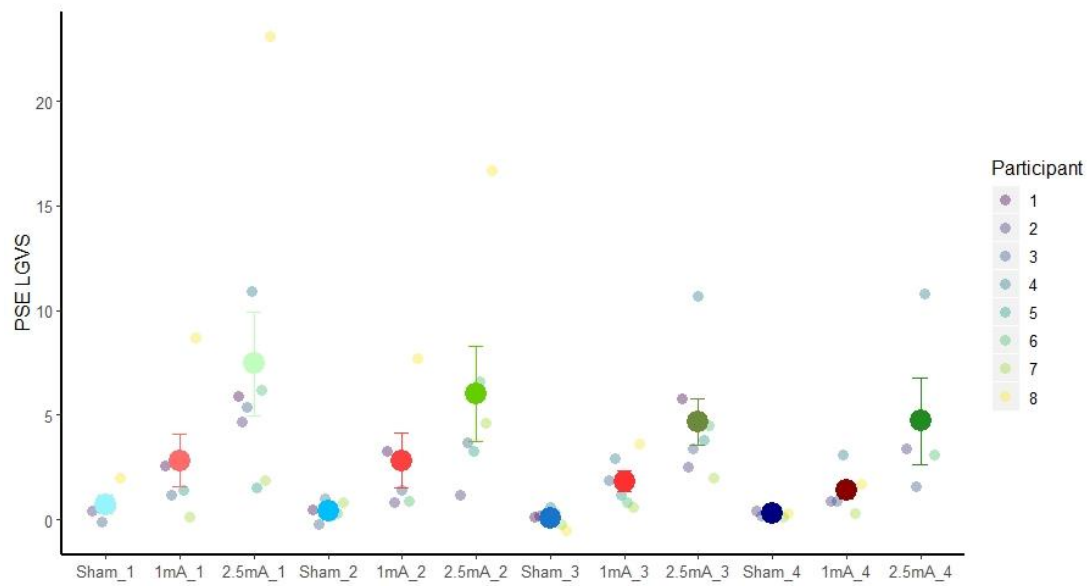
Figure A20. Chapter 8, Left-Anodal/Right-Cathodal GVS Point of Subjective Equality. PSEs for each LGVS Amplitude (Sham, 1mA, 2.5mA) and Session (1-4). Significant differences were found between GVS Amplitudes, with higher PSEs for increasing GVS amplitudes, while there was no effect of Session. Positive numbers represent perceptions of roll to the right. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
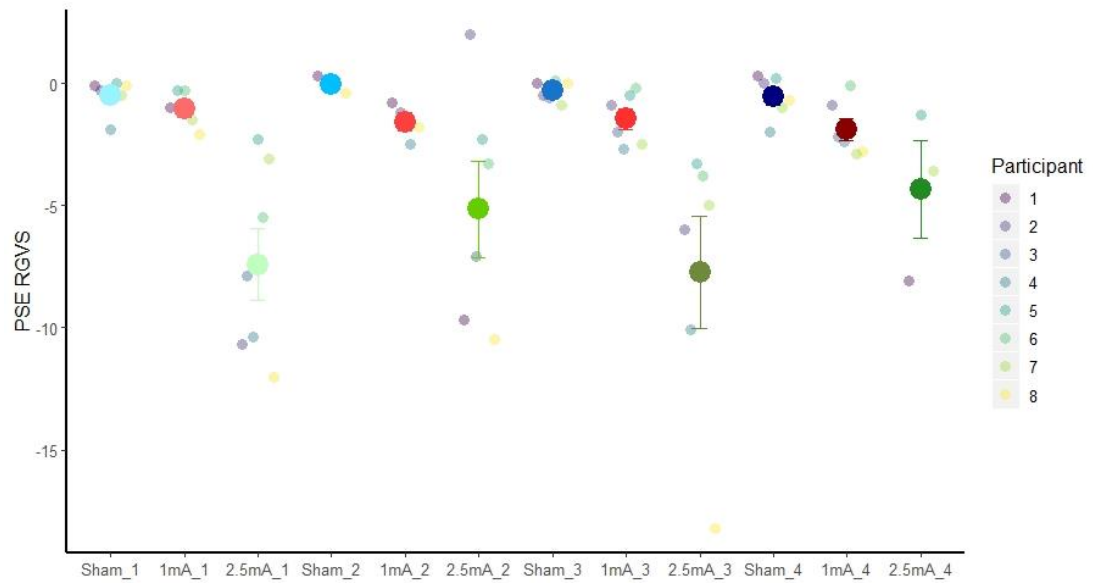
Figure A21. Chapter 8, Right-Anodal/Left-Cathodal GVS Point of Subjective Equality. PSEs for each RGVS Amplitude (Sham, 1mA, 2.5mA) and Session (1-4). Significant differences were found between GVS Amplitudes, with higher PSEs for increasing GVS amplitudes, while there was no effect of Session. Negative numbers represent perceptions of roll to the left. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
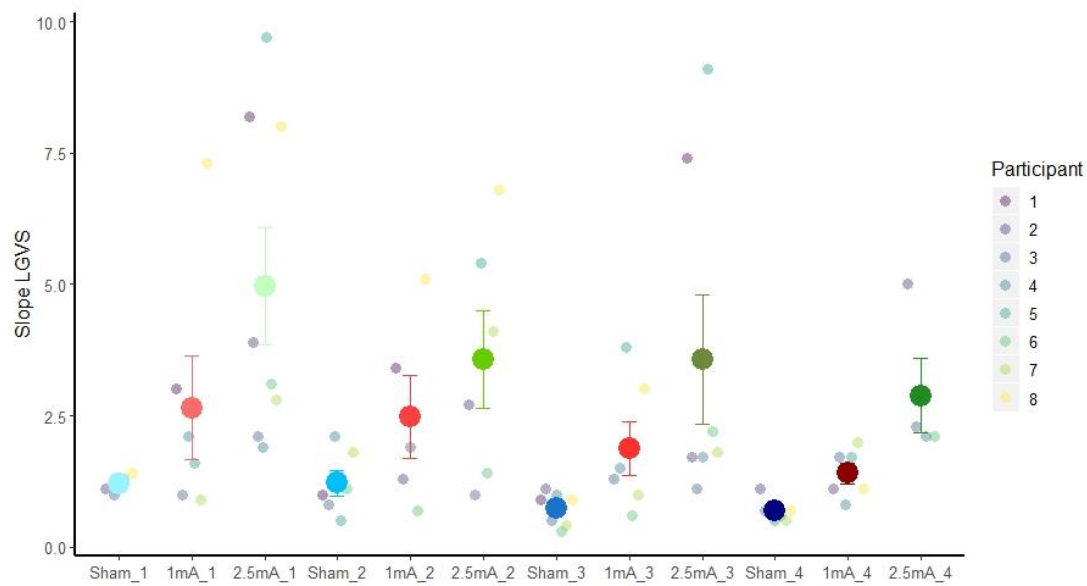
Figure A22. Chapter 8, Left-Anodal/Right-Cathodal GVS Slopes. Slopes for each LGVS Amplitude (Sham, 1mA, 2.5mA) and Session (1-4). Significant differences were found between GVS Amplitudes, with higher slopes (i.e., lower precision) for increasing GVS amplitudes, while there was no effect of Session. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.
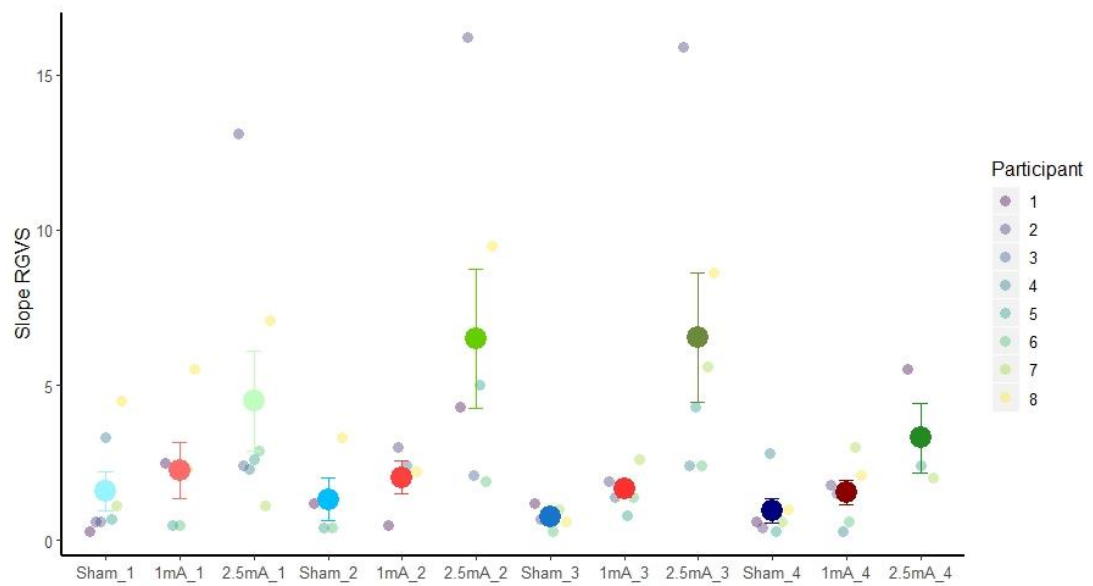
Figure A23. Chapter 8, Right-Anodal/Left-Cathodal GVS Slopes. Slopes for each RGVS Amplitude (Sham, 1mA, 2.5mA) and Session (1-4). Significant differences were found between GVS Amplitudes, with higher slopes (i.e., lower precision) for increasing GVS amplitudes, while there was no effect of Session. Small dots show individual participant results, while large dots indicate the group mean. Error bars represent standard deviation.

Appendix 5: Experimental Data

Chapter 4: Experimental Data

Key to Table A1 Data

- Participant – Participant ID
- Exclude
    - 0 = Participant included in analysis
    - 1 = Participant excluded from analysis
- d
    - d' value for each GVS polarity, the average of both polarities, and visual condition
    - Index = Optic flow condition – Random motion condition
- C
    - Criterion for each GVS polarity, the average of both polarities, and visual condition
    - Index = Optic flow condition – Random motion condition

Table A1. Chapter 4, Experiment 1 d' and Criterion Data

Participant	Exclude	d RGVS Random	d RGVS Flow	d LGVS Random	d LGVS Flow	d Average Random	d Average Flow	d Index	C LGVS Random	C LGVS Flow	C RGVS Random	C RGVS Flow	C Average Flow	C Average Flow	C Index
1	0	2.4568	1.8418	3.0955	1.9176	1.8797	2.7762	-0.8965	0.5803	0.8751	0.6055	0.5802	0.7277	0.5929	0.1348
2	0	0.9409	0.4746	0.2195	0.1434	0.3090	0.5802	-0.2712	1.3913	1.0391	0.8111	0.4906	0.7649	1.1012	-0.3364
3	1	4.2561	3.9620	3.6291	3.2388	3.6004	3.9426	-0.3422	0.3135	0.5086	0.0000	0.1471	0.3279	0.1567	0.1711
4	0	0.5067	0.3328	0.7580	-0.3328	0.0000	0.6323	-0.6323	0.4626	-1.6675	0.0000	-1.6675	-1.6675	0.2313	-1.8988
5	0	3.4096	0.7580	2.8560	1.0488	0.9034	3.1328	-2.2294	0.7001	0.0000	0.4232	0.4626	0.2313	0.5617	-0.3303
6	0	1.3095	1.8418	0.6595	1.5011	1.6714	0.9845	0.6869	1.1714	0.7505	-1.1792	-0.5802	0.0852	-0.0039	0.0891
7	0	1.6222	0.8782	0.9636	0.9429	0.9105	1.2929	-0.3824	-0.1411	-0.6393	0.4704	1.0620	0.2113	0.1647	0.0467
8	0	1.1604	0.2195	1.3332	0.7701	0.4948	1.2468	-0.7520	0.8345	0.7257	0.9209	1.3913	1.0585	0.8777	0.1808
9	0	0.2515	-0.5144	0.9813	-0.7777	-0.6461	0.6164	-1.2625	0.2373	0.1355	-0.0421	0.1735	0.1545	0.0976	0.0570
10	0	2.7826	0.5337	2.3923	0.0000	0.2668	2.5875	-2.3206	0.0854	1.2816	-0.1098	1.2343	1.2579	-0.0122	1.2701
11	0	2.5588	0.5986	1.5695	1.0095	0.8041	2.0642	-1.2601	0.0569	0.3369	0.8487	0.1314	0.2341	0.4528	-0.2186
12	0	2.6524	0.5864	2.7510	0.4430	0.5147	2.7017	-2.1870	0.7526	0.7459	0.8018	0.8176	0.7817	0.7772	0.0046
13	0	0.2533	0.1673	0.0000	-0.0985	0.0344	0.1267	-0.0923	0.2533	0.5737	0.1267	0.0000	0.2868	0.1900	0.0968
14	1	1.7337	2.7826	2.7510	3.4096	3.0961	2.2423	0.8538	0.7526	0.4232	0.2439	-0.1098	0.1567	0.4982	-0.3415
15	0	1.8339	0.3141	2.0444	-0.2692	0.0225	1.9392	-1.9167	1.1058	0.9762	0.9170	1.1245	1.0503	1.0114	0.0389
16	0	0.5524	0.0000	2.8560	2.9697	1.4848	1.7042	-0.2193	-0.7001	-0.6432	1.5577	1.1108	0.2338	0.4288	-0.1951
17	0	0.7714	1.3660	1.3981	0.5144	0.9402	1.0848	-0.1446	0.2683	0.1735	-0.0450	-0.1586	0.0075	0.1117	-0.1042
18	0	1.6352	0.1728	1.6222	0.2570	0.2149	1.6287	-1.4138	0.4704	0.2122	0.2932	0.2543	0.2332	0.3818	-0.1486
19	0	3.6291	1.8387	4.2561	1.9045	1.8716	3.9426	-2.0710	0.0000	0.3293	-0.3135	0.1914	0.2604	-0.1567	0.4171
20	0	0.8465	0.0000	0.5524	-0.0855	-0.0427	0.6994	-0.7422	1.5577	0.2106	1.7048	0.1679	0.1893	1.6313	-1.4420
21	0	1.8060	0.4550	3.0955	0.2515	0.3533	2.4507	-2.0974	0.5803	0.0421	0.3786	0.3954	0.2188	0.4794	-0.2607
22	0	2.7510	1.0704	2.2959	0.7231	0.8968	2.5235	-1.6267	0.9801	1.4723	0.7526	0.9659	1.2191	0.8663	0.3528
23	0	3.9620	0.5524	3.9620	1.2864	0.9194	3.9620	-3.0426	0.1471	1.4848	0.1471	1.5577	1.5213	0.1471	1.3742
24	0	2.4685	1.4494	2.8560	1.1353	1.2924	2.6622	-1.3699	0.7001	0.3998	0.2668	0.5568	0.4783	0.4834	-0.0052

Key to Table A2 Data

- Participant – Participant ID
- Exclude
    - 0 = Participant included in analysis
    - 1 = Participant excluded from analysis
- H – Hit
- FA – False Alarm
- M – Miss
- CR – Correct Reject

Table A2: Chapter 4, Experiment 1, Hits, Misses, False Alarms and Correct Rejects

Participant	Exclude	RGVS								LGVS								Average							
		Optic Flow				Random Motion				Optic Flow				Random Motion				Optic Flow				Random Motion			
		H	FA	M	CR	H	FA	M	CR	H	FA	M	CR	H	FA	M	CR	H	FA	M	CR	H	FA	M	CR
1	0	19	2	11	28	22	1	8	29	16	1	14	29	25	0	5	30	35	3	25	57	47	1	13	59
2	0	12	7	18	23	11	3	19	27	5	4	25	26	3	2	27	28	17	11	43	49	14	5	46	55
3	1	29	0	1	30	30	0	0	30	26	0	4	30	28	0	2	30	55	0	5	60	58	0	2	60
4	0	29	28	1	2	18	12	12	18	28	29	2	1	14	6	16	24	57	57	3	3	32	18	28	42
5	0	14	6	16	24	27	0	3	30	21	9	9	21	23	0	7	30	35	15	25	45	50	0	10	60
6	0	28	11	2	19	29	21	1	9	15	2	15	28	6	2	24	28	43	13	17	47	35	23	25	37
7	0	8	2	22	28	19	3	11	27	26	17	4	13	22	11	8	19	34	19	26	41	41	14	19	46
8	0	3	2	27	28	11	2	19	28	11	4	19	26	13	2	17	28	14	6	46	54	24	4	36	56
9	0	10	16	20	14	17	14	13	16	9	18	21	12	18	7	12	23	19	34	41	26	35	21	25	39
10	0	5	2	25	28	28	3	2	27	3	3	27	27	26	3	4	27	8	5	52	55	54	6	6	54
11	0	17	10	13	20	20	0	10	30	17	6	13	24	23	6	7	24	34	16	26	44	43	6	17	54
12	0	9	4	21	26	21	0	9	30	9	5	21	25	22	0	8	30	18	9	42	51	43	0	17	60
13	0	16	14	14	16	15	12	15	18	8	9	22	21	12	12	18	18	24	23	36	37	27	24	33	36
14	1	28	3	2	27	22	4	8	26	27	0	3	30	22	0	8	30	55	3	5	57	44	4	16	56
15	0	5	3	25	27	15	1	15	29	4	6	26	24	14	0	16	30	9	9	51	51	29	1	31	59
16	0	4	4	26	26	3	1	27	29	30	6	0	24	30	7	0	23	34	10	26	50	33	8	27	52
17	0	24	9	6	21	20	11	10	19	16	10	14	20	20	5	10	25	40	19	20	41	40	16	20	44
18	0	13	11	17	19	21	4	9	26	14	11	16	19	19	3	11	27	27	22	33	38	40	7	20	53
19	0	23	4	7	26	30	2	0	28	22	3	8	27	30	0	0	30	45	7	15	53	60	2	0	58
20	0	13	13	17	17	3	0	27	30	12	13	18	17	3	1	27	29	25	26	35	34	6	1	54	59
21	0	13	8	17	22	21	3	9	27	16	13	14	17	25	0	5	30	29	21	31	39	46	3	14	57
22	0	10	2	20	28	22	0	8	30	4	1	26	29	17	0	13	30	14	3	46	57	39	0	21	60
23	0	3	1	27	29	29	0	1	30	6	0	24	30	29	0	1	30	9	1	51	59	58	0	2	60
24	0	17	3	13	27	25	2	5	28	17	5	13	25	23	0	7	30	34	8	26	52	48	2	12	58

Key to Table A3 Data

- Participant – Participant ID
- Exclude
    - 0 = Participant included in analysis
    - 1 = Participant excluded from analysis
- d
    - d' value for each GVS polarity, the average of both polarities, and visual condition
    - Index = Optic flow condition – Random motion condition
- C
    - Criterion for each GVS polarity, the average of both polarities, and visual condition
    - Index = Optic flow condition – Random motion condition

Table A3. Chapter 4, Experiment 2 d' and Criterion Data

Participant	Exclude	d RGVS Random	d RGVS Flow	d LGVS Random	d LGVS Flow	d Average Random	d Average Flow	d Index	C LGVS Random	C LGVS Flow	C RGVS Random	C RGVS Flow	C Average Flow	C Average Flow	C Index
1	0	0.2941	1.6973	1.4932	2.4568	2.0771	0.8937	1.1834	1.0873	0.6055	1.9810	1.2794	0.9424	1.5341	-0.5917
2	0	3.9620	3.3350	1.1944	2.5618	2.9484	2.5782	0.3702	0.5136	0.5530	0.1471	-0.1664	0.1933	0.3303	-0.1370
3	0	-0.3903	1.0271	0.1137	1.1604	1.0938	-0.1383	1.2321	0.7848	0.9209	1.3059	0.5972	0.7591	1.0453	-0.2863
4	1	3.6678	2.0873	4.2561	2.0018	2.0445	3.9620	-1.9174	0.0000	0.8330	0.0000	0.7903	0.8116	0.0000	0.8116
5	0	1.7503	0.6841	0.9674	1.6832	1.1837	1.3588	-0.1752	0.4837	0.0000	0.9588	0.0887	0.0443	0.7212	-0.6769
6	0	2.2959	2.7510	2.3814	2.4687	2.6099	2.3387	0.2712	0.9373	0.8937	0.9801	0.7526	0.8231	0.9587	-0.1356
7	0	1.6352	1.9176	1.0537	2.4568	2.1872	1.3444	0.8428	0.0961	0.6055	-0.2932	0.8751	0.7403	-0.0985	0.8389
8	0	1.6660	1.9524	1.5051	1.5903	1.7714	1.5856	0.1858	1.3755	0.1722	1.0009	0.1346	0.1534	1.1882	-1.0348
9	0	1.1137	-0.0900	0.2187	-0.1922	-0.1411	0.6662	-0.8073	0.7323	0.5268	0.7247	0.3857	0.4563	0.7285	-0.2722
10	0	1.8387	0.6229	2.4685	1.7337	1.1783	2.1536	-0.9753	-0.2668	0.2439	-0.1914	0.3115	0.2777	-0.2291	0.5068
11	1	3.9620	2.9697	3.9620	3.0955	3.0326	3.9620	-0.9294	0.1471	0.5803	0.1471	0.6432	0.6118	0.1471	0.4647
12	0	2.3814	1.6660	1.6660	1.4032	1.5346	2.0237	-0.4891	1.0009	1.1323	0.9373	1.0009	1.0666	0.9691	0.0975
13	0	0.8574	-0.0873	0.5367	-0.4407	-0.2640	0.6971	-0.9611	0.6991	-0.3040	0.6821	-0.2970	-0.3005	0.6906	-0.9911
14	0	2.7826	3.3350	3.1155	3.6678	3.5014	2.9491	0.5524	-0.2762	0.0000	-0.1098	-0.1664	-0.0832	-0.1930	0.1098
15	0	1.2787	0.6270	1.1108	1.2864	0.9567	1.1947	-0.2380	0.5554	1.4848	0.4714	1.8146	1.6497	0.5134	1.1363
16	0	2.6755	1.4001	3.2388	2.3814	1.8908	2.9572	-1.0664	0.5086	0.9373	0.4961	1.4280	1.1827	0.5024	0.6803
17	0	1.7123	2.0018	1.7544	1.2208	1.6113	1.7334	-0.1221	0.6239	0.3570	0.4254	0.8330	0.5950	0.5246	0.0704
18	0	2.8560	1.5847	2.3583	2.7510	2.1679	2.6071	-0.4393	0.6548	0.7526	0.7001	0.7087	0.7306	0.6774	0.0532
19	0	1.0095	-0.2035	0.0000	0.3471	0.0718	0.5048	-0.4330	-0.4307	0.2572	-0.3369	0.6262	0.4417	-0.3838	0.8255
20	0	1.4515	0.9429	0.8574	0.4746	0.7087	1.1544	-0.4457	0.6821	0.4906	0.3850	0.6393	0.5650	0.5335	0.0314
21	0	1.5051	2.5588	1.5051	2.4687	2.5138	1.5051	1.0086	1.3755	0.8937	1.3755	0.8487	0.8712	1.3755	-0.5043
22	0	0.9253	0.5883	1.4645	0.1837	0.3860	1.1949	-0.8089	0.1093	0.4325	0.3790	0.5475	0.4900	0.2442	0.2458
23	0	-0.1697	0.5367	0.4407	0.8574	0.6971	0.1355	0.5615	0.3040	0.6821	0.1685	0.6991	0.6906	0.2363	0.4543
24	0	0.8782	0.2941	1.9176	0.8782	0.5861	1.3979	-0.8117	0.8751	1.0620	1.0620	1.9810	1.5215	0.9686	0.5529

Key to Table A4 Data

- Participant – Participant ID
- Exclude
    - 0 = Participant included in analysis
    - 1 = Participant excluded from analysis
- H – Hit
- FA – False Alarm
- M – Miss
- CR – Correct Reject

Table A4. Chapter 4, Experiment 2 Hits, False Alarms, Misses, Correct Rejects

Participant	Exclude	RGVS								LGVS								Average							
		Optic Flow				Random Motion				Optic Flow				Random Motion				Optic Flow				Random Motion			
		H	FA	M	CR	H	FA	M	CR	H	FA	M	CR	H	FA	M	CR	H	FA	M	CR	H	FA	M	CR
1	0	10	0	20	30	1	0	29	30	22	1	8	29	11	1	19	29	32	1	28	59	12	1	48	59
2	0	29	2	1	28	29	0	1	30	23	1	7	29	16	4	14	26	52	3	8	57	45	4	15	56
3	0	14	4	16	26	2	4	28	26	11	2	19	28	7	6	23	24	25	6	35	54	9	10	51	50
4	1	18	1	12	29	29	1	1	29	17	1	13	29	30	0	0	30	35	2	25	58	59	1	1	59
5	0	18	10	12	20	14	1	16	29	24	6	6	24	15	5	15	25	42	16	18	44	29	6	31	54
6	0	22	0	8	30	17	0	13	30	19	0	11	30	18	0	12	30	41	0	19	60	35	0	25	60
7	0	16	1	14	29	26	9	4	21	22	1	8	29	20	8	10	22	38	2	22	58	46	17	14	43
8	0	24	4	6	26	13	1	17	29	22	5	8	25	8	0	22	30	46	9	14	51	21	1	39	59
9	0	10	11	20	19	13	3	17	27	8	10	22	20	8	6	22	24	18	21	42	39	21	9	39	51
10	0	15	8	15	22	26	7	4	23	22	4	8	26	28	5	2	25	37	12	23	48	54	12	6	48
11	1	24	0	6	30	29	0	1	30	25	0	5	30	29	0	1	30	49	0	11	60	58	0	2	60
12	0	13	1	17	29	18	0	12	30	10	1	20	29	13	1	17	29	23	2	37	58	31	1	29	59
13	0	18	19	12	11	12	4	18	26	16	21	14	9	10	5	20	25	34	40	26	20	22	9	38	51
14	0	29	2	1	28	28	3	2	27	29	1	1	29	29	3	1	27	58	3	2	57	57	6	3	54
15	0	2	0	28	30	17	4	13	26	6	0	24	30	15	4	15	26	8	0	52	60	32	8	28	52
16	0	7	0	23	30	24	1	6	29	18	0	12	30	26	0	4	30	25	0	35	60	50	1	10	59
17	0	17	1	13	29	20	3	10	27	18	5	12	25	18	2	12	28	35	6	25	54	38	5	22	55
18	0	16	2	14	28	23	0	7	30	22	0	8	30	21	1	9	29	38	2	22	58	44	1	16	59
19	0	7	9	23	21	24	13	6	17	14	10	16	20	20	20	10	10	21	19	39	41	44	33	16	27
20	0	13	4	17	26	19	4	11	26	12	7	18	23	12	4	18	26	25	11	35	49	31	8	29	52
21	0	20	0	10	30	8	0	22	30	19	0	11	30	8	0	22	30	39	0	21	60	16	0	44	60
22	0	12	6	18	24	16	6	14	24	11	9	19	21	22	6	8	24	23	15	37	45	38	12	22	48
23	0	10	5	20	25	12	14	18	16	12	4	18	26	14	9	16	21	22	9	38	51	26	23	34	37
24	0	1	0	29	30	8	2	22	28	8	2	22	28	16	1	14	29	9	2	51	58	24	3	36	57

Chapter 5: Experimental Data

Key to Table A5 Data

- Participant – Participant ID
- Amp – VEMPs P1-N1 Peak-To-Peak amplitude for Vection and Random motion conditions on Left and Right muscle sides
- P1 – VEMPs P1 Latency for Vection and Random motion conditions on Left and Right muscle sides
- N1 – VEMPs N1 Latency for Vection and Random motion conditions on Left and Right muscle sides
- Asym – VEMPs Asymmetry ratio for Vection and Random motion conditions
- Int – VEMPs P1-N1 latency interval for Vection and Random motion conditions on Left and Right muscle sides
- MSSQ Percent – Motion Sickness Susceptibility Questionnaire Percentile scores

Table A5: Chapter 5 VEMPs data.

Participant	Amp Vection Left	Amp Random Left	Amp Vection Right	Amp Random Right	P1 Vection Left	P1 Random Left	P1 Vection Right	P1 Random Right	N1 Vection Left	N1 Random Left	N1 Vection Right	N1 Random Right	Asym Vection	Asym Random	Int Vection Left	Int Random Left	Int Vection Right	Int Random Right	MSSQ Percent
1	311.8	227.4	294.4	247.5	17.5	12.9	12.8	12.3	27.8	23.8	21.9	23.9	-2.87	4.23	10.3	10.9	9.1	11.6	65.56
2	227.9	250.6	274	263.8	11.3	12.1	12.2	12.3	21.2	27.2	24.3	29.9	9.19	2.57	9.9	15.1	12.1	17.6	33.82
3	217.1	213	188	287.5	22.5	19.9	20.9	14.4	31.3	27.1	31	26.4	-7.18	14.89	8.8	7.2	10.1	12	19.54
4	234	236.3	246.1	276.8	11.7	11.6	9.2	12.9	21.3	21	22.2	25	2.52	7.89	9.6	9.4	13	12.1	5.06
5	467.6	446	276.3	252	16.7	12.3	11.8	12.9	27.2	27.8	25.3	24.1	-25.72	-27.79	10.5	15.5	13.5	11.2	0
6	326	279.5	219.7	231.2	9.9	11.5	7.4	7.4	21	29.8	25.8	29.9	-19.48	-9.46	11.1	18.3	18.4	22.5	77.24
7	258.1	174.3	278.3	257.8	12.6	11	10.7	20.7	20.4	27.7	19.8	28	3.77	19.32	7.8	16.7	9.1	7.3	47.82
8	177.6	197.6	187.2	171.3	21.7	8.6	17	20.4	29.1	23.1	22.1	25.9	2.63	-7.13	7.4	14.5	5.1	5.5	37.01
9	244.4	280.1	274	196.8	10.5	22.8	21.2	15	28.1	31.2	29.4	33.1	5.71	-17.47	17.6	8.4	8.2	18.1	19.54
10	390	346	189	289.2	18	19.3	20.3	9.3	28.2	29.7	30.7	20.3	-34.72	-8.94	10.2	10.4	10.4	11	42.96
11	428.9	316.5	343.7	222.8	14.7	14.7	9.3	22.9	32.9	22.7	33.4	29.9	-11.03	-17.37	18.2	8	24.1	7	64.26
12	368.5	338.8	302.2	346.8	14.6	15.5	9.1	16	31.1	27.8	24.4	29.9	-9.89	1.17	16.5	12.3	15.3	13.9	41.01
13	337.7	260.3	436.7	275.3	15.6	16.9	16.6	17.3	25.4	28.9	26.1	25.2	12.78	2.8	9.8	12	9.5	7.9	21.84
14	202.7	201.2	232.3	266.5	11.7	12.1	11.4	11.1	21.1	21.1	21.1	21.1	6.8	13.96	9.4	9	9.7	10	17.53
15	224.8	207.8	342.4	272.9	14.4	16.9	14.5	13.7	24.1	32.2	22.8	22.5	20.73	13.54	9.7	15.3	8.3	8.8	77.24
16	242.3	293.4	243.9	326.5	15.9	15.4	10.6	16	27.8	26	30	28.6	0.33	5.34	11.9	10.6	19.4	12.6	28.55
17	380.2	255.9	275.4	278.6	23.1	11.8	20.5	20	34	23.7	33.1	33.4	-15.99	4.25	10.9	11.9	12.6	13.4	0
18	252.5	270.2	234.9	245	16.1	15.1	11.3	17.9	29.6	25.2	20	25.5	-3.61	-4.89	13.5	10.1	8.7	7.6	77.24
19	208.8	234.6	119.2	204.9	16.8	14.6	11.9	21.4	30.3	32.4	21.7	33.4	-27.32	-6.76	13.5	17.8	9.8	12	0
20	198.4	210	211.8	224.5	8	8.9	19.2	22.9	25.8	32.1	29	33.3	3.27	3.34	17.8	23.2	9.8	10.4	64.26
21	268.8	212.2	199.2	227.4	19.5	14.6	9.2	9.9	27.8	33.9	22	32	-14.87	3.46	8.3	19.3	12.8	22.1	0
22	303.2	190.8	254.2	291.5	19.3	15.5	22.8	16.7	28.3	32.7	33.7	25	-8.79	20.88	9	17.2	10.9	8.3	76.39
23	178.2	173.7	277.6	261.2	22.2	21.6	18.6	18.6	31.4	29.5	27	30.3	21.81	20.12	9.2	7.9	8.4	11.7	74.75
24	305.8	241.8	219.9	202.9	14.9	19.2	12.5	11.5	27.1	27.9	27.8	20.9	-16.34	-8.75	12.2	8.7	15.3	9.4	66.8

Chapter 6: Experimental Data

Key to Table A6 Data:

- Participant – Participant ID
- Condition
    - 0 = Upright
    - 1 = Tilted
- SSQ – Simulator Sickness Questionnaire Scores
    - T = Total Score
    - N = Nausea Subscale Score
    - O = Oculomotor Subscale Score
    - D = Disorientation Subscale Score

Table A6: Chapter 6, Simulator Sickness Questionnaire Data

Participant	Condition	SSQ T	SSQ N	SSQ O	SSQ D
1	0	29.92	19.08	22.74	41.76
2	1	26.18	36.16	7.58	27.84
3	0	29.92	28.62	22.74	27.84
4	1	22.44	28.62	7.58	27.84
5	0	71.06	47.7	60.64	83.52
6	1	97.24	47.7	83.38	139.2
7	0	22.44	28.62	22.74	0
8	1	78.54	76.32	30.32	125.28
9	0	56.1	38.16	53.06	55.68
10	1	29.92	38.16	15.16	27.84
11	0	44.88	57.24	22.74	41.76
12	1	22.44	38.16	15.16	0
13	1	63.58	47.7	53.06	69.6
14	0	29.92	38.16	15.16	27.84
15	1	71.06	57.24	53.06	83.52
16	0	187	133.56	144.02	236.64
17	1	67.32	57.24	37.9	97.44
18	0	63.58	76.32	22.74	83.52
19	1	41.14	47.7	22.74	41.76
20	0	44.88	28.62	22.74	83.52
21	1	22.44	9.54	22.74	27.84
22	0	59.84	66.78	30.32	69.6
23	1	18.7	28.62	7.58	13.92
24	0	48.62	47.7	15.16	83.52

Key to Table A7 Data:

- Participant – Participant ID
- Condition
    - 0 = Upright
    - 1 = Tilted
- FMS – Raw Fast Motion Sickness Scores
    - Pre = Pre-VR Exposure
    - 1-10 = During-VR Exposure
    - Post = Post-VR Exposure
    - Peak = Peak During-VR Score
    - Slope = Linear Regression Slope During-VR

Table A7: Chapter 6, Raw Fast Motion Sickness Scale Data

Participant	Condition	FMS Pre	FMS 1	FMS 2	FMS 3	FMS 4	FMS 5	FMS 6	FMS 7	FMS 8	FMS 9	FMS 10	FMS Post	FMS Peak	FMS Slope
1	0	0	0	0	0	2	1	2	2	2	2	1	1	2	2.237
2	1	0	0	1	1	4	4	4	4	4	5	4	5	5	1.468
3	0	0	0	1	1	1	2	2	2	1	1	1	1	2	1.667
4	1	0	2	3	4	4	4	3	4	5	5	6	5	6	2.250
5	0	0	6	10	14	15	15	16	15	15	15	16	14	16	0.711
6	1	0	5	5	4	6	7	8	8	9	8	9	9	9	1.540
7	0	0	4	3	5	5	6	6	5	6	7	7	6	7	2.083
8	1	0	5	12	15	15	12	15	14	14	10	8	5	15	0.038
9	0	0	2	4	4	4	4	5	4	5	6	6	5	6	2.258
10	1	0	8	6	8	7	3	0	0	3	0	3	1	8	-0.711
11	0	1	5	4	6	11	8	8	11	10	10	5	5	11	0.487
12	1	0	0	0	1	1	3	5	4	4	2	1	0	5	0.848
13	1	1	2	2	2	3	5	6	6	6	7	7	6	7	1.361
14	0	0	4	5	6	7	7	7	8	8	8	8	8	8	1.989
15	1	0	3	2	3	2	2	1	4	8	9	9	10	9	0.755
16	0	0	7	10	10	12	13	14	15	16	16	16	17	16	0.938
17	1	4	4	7	8	7	6	6	5	5	5	5	4	8	-0.956
18	0	0	7	10	10	12	14	16	16	8	18	18	18	18	0.534
19	1	1	4	6	7	7	9	11	10	10	9	12	9	12	1.092
20	0	1	6	5	7	6	4	5	4	4	3	3	4	7	-1.894
21	1	2	5	5	5	7	7	9	11	9	9	8	8	11	1.156
22	0	0	0	0	0	1	1	1	2	2	3	3	3	3	2.521
23	1	0	0	2	3	3	7	9	9	11	11	12	9	12	0.679
24	0	0	3	4	7	10	10	12	14	15	14	16	17	16	0.639

Key to Table A8 Data:

- Participant – Participant ID
- Condition
  - 0 = Upright
  - 1 = Tilted
- HR – Raw Heart Rate
  - Pre = Pre-VR Exposure
  - 1-10 = During-VR Exposure
  - Post = Post-VR Exposure
  - Peak = Peak During-VR Score
  - Slope = Linear Regression Slope During-VR

Table A8: Chapter 6, Raw Heart Rate Data

Participant	Condition	HR Pre	HR 1	HR 2	HR 3	HR 4	HR 5	HR 6	HR 7	HR 8	HR 9	HR 10	HR Post	HR Peak	HR Slope
1	0	90	93	93	91	94	96	93	96	109	97	99	102	109	0.372
2	1	104	102	106	109	117	111	115	117	115	110	111	110	117	0.334
3	0	135	124	122	116	127	114	115	117	112	118	113	111	127	-0.394
4	1	81	79	90	81	81	84	85	91	94	89	92	90	94	0.411
5	0	78	71	80	74	79	74	82	77	76	77	80	77	82	0.353
6	1	95	91	91	97	90	96	98	109	98	102	104	91	109	0.381
7	0	82	68	75	67	79	81	79	81	83	85	87	77	87	0.404
8	1	80	76	81	78	78	79	78	78	84	81	83	79	84	0.788
9	0	75	81	74	76	74	73	72	84	83	81	79	71	84	0.270
10	1	83	80	81	81	85	82	85	88	84	81	87	87	88	0.639
11	0	96	98	98	97	95	90	98	96	94	95	93	89	98	-0.579
12	1	89	88	90	93	97	97	92	95	96	97	94	89	97	0.602
13	1	85	82	82	84	85	85	88	89	89	88	86	77	89	0.928
14	0	92	90	91	92	93	88	90	91	90	89	89	94	93	-0.920
15	1	72	73	72	75	73	75	76	76	77	77	83	70	83	0.842
16	0	98	95	99	109	104	109	107	115	117	99	115	102	117	0.243
17	1	89	98	87	98	96	107	93	95	96	91	96	97	107	-0.035
18	0	114	111	110	117	110	116	116	111	114	115	116	113	117	0.483
19	1	96	94	91	98	101	93	102	100	97	95	102	103	102	0.360
20	0	74	73	73	77	80	81	81	81	82	81	81	81	82	0.736
21	1	77	77	75	79	80	81	80	80	77	82	84	80	84	0.808
22	0	92	90	96	92	90	93	90	94	88	88	89	88	96	-0.594
23	1	83	82	80	84	87	87	92	93	95	94	94	90	95	0.519
24	0	80	74	76	77	85	80	80	82	86	85	90	85	90	0.532

Chapter 7: Experimental Data

Key to Table A9 Data:

- Participant – Participant ID
- Order – Order conditions were presented to participants
  - 0 = GVS First
  - 1 = Sham First
- Exclude – Participants to be excluded from analysis
  - 0 = Included in Analysis
  - 1 = Excluded from Analysis
- SSQ – Simulator Sickness Questionnaire Scores for GVS and Sham conditions
  - T = Total Score
  - N = Nausea Subscale Score
  - D = Disorientation Subscale Score
  - O = Oculomotor Subscale Score

Table A9: Chapter 7, Simulator Sickness Questionnaire Data

Participant	Order	Exclude	SSQ T GVS	SSQ N GVS	SSQ D GVS	SSQ O GVS	SSQ T Sham	SSQ N Sham	SSQ D Sham	SSQ O Sham
1	0	0	33.66	19.08	27.84	37.9	26.18	19.08	27.84	37.9
2	1	0	26.18	9.54	27.84	37.9	22.44	19.08	41.76	22.74
3	0	0	26.18	19.08	41.76	53.06	14.96	9.54	27.84	22.74
4	1	0	22.44	19.08	27.84	22.74	37.4	28.62	83.52	22.74
5	0	0	29.92	28.62	69.6	30.32	26.18	9.54	55.68	53.06
6	1	0	11.22	9.54	22.74	13.92	18.7	28.02	13.92	30.32
7	0	0	29.92	19.08	55.68	37.9	0	0	0	0
8	1	0	0	0	0	0	0	0	0	0
9	0	0	67.32	38.16	53.06	69.6	63.58	47.7	97.44	90.96
10	1	0	0	0	0	0	7.48	0	13.92	15.16
11	0	1	56.1	85.86	83.52	60.64	22.44	28.62	27.84	22.74
12	1	0	18.7	9.54	13.92	37.9	29.92	9.54	41.76	53.06
13	0	0	7.48	0	27.84	0	3.74	0	0	7.58
14	1	1	7.48	0	13.92	15.16	22.44	28.62	27.84	37.9
15	0	0	0	0	0	0	0	0	0	0

Key to Table A10 Data:

- Participant – Participant ID
- Order – Order conditions were presented to participants
  - 0 = GVS First
  - 1 = Sham First
- Exclude – Participants to be excluded from analysis
  - 0 = Included in Analysis
  - 1 = Excluded from Analysis
- Asymm – VEMPs Asymmetry Ratios for GVS and Sham Conditions Pre- and Post-VR

Table A10: Chapter 7, VEMPs Asymmetry Data

Participant	Order	Exclude	Asym Pre GVS	Asym Post GVS	Asym Pre Sham	Asym Post Sham
1	0	0	-0.4	10.3	19.1	25.5
2	1	0	-8.6	-16.4	-1.8	-14.9
3	0	0	-10.7	26.8	7.6	8.8
4	1	0	-6.6	8.8	-1	-1.1
5	0	0	7	3.6	2.8	29.3
6	1	0	11.8	-12.6	1.6	-23.9
7	0	0	-5.5	14.6	-7.8	-0.5
8	1	0	1.7	14.4	4.2	4.8
9	0	0	12.9	8.4	-21.9	4.6
10	1	0	8.5	0.6	-1.4	11.7
11	0	1	-1.3	11.2	8.5	-0.5
12	1	0	-4.6	-1.1	22.2	4.7
13	0	0	10.4	6.3	20.8	3.3
14	1	1	-1.4	-6.5	-1.1	Recording Err.
15	0	0	-14.5	-6.8	-4.6	-1.7

Key to Table A11 Data:

- Participant – Participant ID
- Order – Order conditions were presented to participants
  - 0 = GVS First
  - 1 = Sham First
- Exclude – Participants to be excluded from analysis
  - 0 = Included in Analysis
  - 1 = Excluded from Analysis
- Amp – VEMPs P1-N1 Peak-to-Peak Amplitude Pre- and Post-VR Exposure for Left and Right Muscle Sides, and GVS and Sham conditions

Table A11: Chapter 7, VEMPs Amplitude Data

Participant	Order	Exclude	Amp Pre Left GVS	Amp Post Left GVS	Amp Pre Left Sham	Amp Post Left Sham	Amp Pre Right GVS	Amp Post Right GVS	Amp Pre Right Sham	Amp Post Right Sham
1	0	0	214.6	175.5	126.8	118	212.9	215.8	186.6	198.7
2	1	0	166.9	168.5	147.3	203.8	140.5	121.1	142	151
3	0	0	214.1	127.1	190.8	173.8	172.8	220	222.1	207.1
4	1	0	266.8	221.5	249.6	261	233.7	261.4	244.7	255.6
5	0	0	209.8	218.7	182.4	134.3	241.5	235	193.1	245.5
6	1	0	189.7	264.3	214.6	230	240.5	205.1	221.5	141.2
7	0	0	224	178.5	297.6	213.7	200.7	239.6	254.6	211.4
8	1	0	226.4	193.8	196.6	243.7	234.1	259.1	213.7	268.6
9	0	0	121.1	163.2	177.4	167.1	156.8	193.2	113.6	183.1
10	1	0	201.8	233.3	211	131.9	239.2	236.1	205.3	167
11	0	1	197	93.4	60.7	48.5	191.9	117.1	72	48.1
12	1	0	269.1	217	143.4	186	245.3	212.4	225.4	204.3
13	0	0	162.2	179.8	194.7	194.2	200	204.1	297	207.2
14	1	1	208.9	51.2	186.6	Recording Err.	203.1	44.9	182.6	172.6
15	0	0	280	289.6	211.8	206.7	209.2	252.8	193	199.7

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Key to Table A12 Data:

- Participant – Participant ID
- Order – Order conditions were presented to participants
  - 0 = GVS First
  - 1 = Sham First
- Exclude – Participants to be excluded from analysis
  - 0 = Included in Analysis
  - 1 = Excluded from Analysis
- P1 – VEMPs P1 Latency Pre- and Post-VR Exposure for Left and Right Muscle Sides, and GVS and Sham conditions

Table A12: Chapter 7, VEMPs P1 Latency Data

Participant	Order	Exclude	P1 Pre Left GVS	P1 Post Left GVS	P1 Pre Left Sham	P1 Post Left Sham	P1 Pre Right GVS	P1 Post Right GVS	P1 Pre Right Sham	P1 Post Right Sham
1	0	0	17.8	16.6	21.9	24	12.7	9.8	16.9	10.9
2	1	0	20.7	20.8	20.5	20.6	11	10.8	19.6	22.6
3	0	0	13.8	12.8	13.7	12.5	7	6.6	12.3	14.1
4	1	0	22.7	16.5	14.4	21.1	12.7	11.6	10.2	9.6
5	0	0	10	23.5	16.1	21.8	15.1	9.5	14.4	16.3
6	1	0	8.2	20.1	18.6	19.8	17.8	12	20.2	17.4
7	0	0	17.9	18.3	19.7	12.4	11.1	14.8	20.1	20.6
8	1	0	14.9	19.8	6.8	5	19.6	19.7	8	20.3
9	0	0	22.1	14.9	11.5	7.3	22.3	19	11.2	20.3
10	1	0	20.1	21.6	21.9	21.8	19.1	17.9	20	20.8
11	0	1	11.9	11.5	26	31.9	10.9	11.5	23.9	17.4
12	1	0	19.9	8.3	10	7.9	20	15.1	19.8	12.7
13	0	0	15	22.7	17.1	9.4	21	10.2	23	16.9
14	1	1	18.3	21.1	10.9	Recording Err.	20.6	15.8	10.2	16.8
15	0	0	16	17.3	11.2	18.4	20.3	17.1	16.6	10.9

Key to Table A13 Data:

- Participant – Participant ID
- Order – Order conditions were presented to participants
  - 0 = GVS First
  - 1 = Sham First
- Exclude – Participants to be excluded from analysis
  - 0 = Included in Analysis
  - 1 = Excluded from Analysis
- N1 – VEMPs N1 Latency Pre- and Post-VR Exposure for Left and Right Muscle Sides, and GVS and Sham conditions

Table A13: Chapter 7, VEMPs N1 Latency Data

Participant	Order	Exclude	N1 Pre Left GVS	N1 Post Left GVS	N1 Pre Left Sham	N1 Post Left Sham	N1 Pre Right GVS	N1 Post Right GVS	N1 Pre Right Sham	N1 Post Right Sham
1	0	0	28.2	31.1	30.3	33.4	24	23.2	25.8	23.6
2	1	0	28.6	28.1	33.8	29.4	29	27.9	27.6	28.9
3	0	0	22.5	22.1	22.3	21.4	21.3	22.8	29	26.7
4	1	0	33.8	26.7	27	32.3	30.5	22.3	23.2	32.8
5	0	0	32.7	34.9	25.3	29.5	26.6	30.9	32.5	26.9
6	1	0	25.8	28.5	27.5	29	26.3	24.7	28.5	23.7
7	0	0	27.3	31.7	30.6	30.5	21.3	33.4	33.6	33.4
8	1	0	27.2	28.4	25.9	28.5	30.4	30.1	28.6	29.6
9	0	0	31.3	26	26.5	21.9	29.1	27.7	27.7	33.2
10	1	0	27.6	33	30.7	29.4	28	29.1	30.3	33.8
11	0	1	33.7	28.1	37.2	40.1	34.5	21.7	34.5	30.5
12	1	0	28.5	26.4	22.1	22.9	29.2	22.3	27.6	26.3
13	0	0	30.7	30.7	24.9	31.8	30	31.1	31.5	29.7
14	1	1	31	32.7	27.1	Recording Err.	31.9	25.9	33.7	25
15	0	0	31.4	29.1	28.8	29.4	29.3	27.8	27.9	31.3

Chapter 8: Experimental Data

Key to Table A14 Data

- Participant – Participant ID
- GVS Amp – GVS Amplitude
    - 0 = Sham stimulation
    - 1 = 1mA GVS
    - 2.5 = 2.5mA GVS
- PSE – Point of Subjective Equality for each GVS polarity and session
    - RGVS = Right-Anodal/Left-Cathodal GVS
    - LGVS = Left-Anodal/Right-Cathodal GVS
    - 1-4 = Session number

Table A14: Chapter 8, Point of Subjective Equality Data

Participant	GVS Amp	PSE LGVS 1	PSE LGVS 2	PSE LGVS 3	PSE LGVS 4	PSE RGVS 1	PSE RGVS 2	PSE RGVS 3	PSE RGVS 4
1	0	0.3	0.5	0.1	1	-0.1	0.3	0	0.3
2	0	0.4	0.1	0.2	0.4	-0.3	0.1	-0.5	0
3	0	-0.1	-0.2	0.1	0.2	-0.4	-1.1	-0.6	-0.6
4	0	-0.6	1	0.6	0.5	-1.9	0.7	-0.6	-2
5	0	-0.4	0.2	0.5	0.2	0	0	1.8	0.2
6	0	0.6	0.3	0.3	0.5	0.1	0	0.1	0.4
7	0	-19.4	0.8	-0.2	0.1	-0.5	-3.4	-0.9	-1
8	0	2	2.4	-0.5	0.3	-0.1	-0.4	0	-0.7
1	1	2.6	3.3	3.4	1.5	-1	-0.8	1.3	-4.9
2	1	0.9	0.8	0.3	0.9	-2.9	-1.2	-0.9	-0.9
3	1	1.2	1.4	1.9	0.9	-2.4	-3	-2	-2.2
4	1	3	3.6	2.9	3.1	-4.7	-2.5	-2.7	-2.4
5	1	1.4	0.5	1.2	1.6	-0.3	0.4	-0.5	-1.6
6	1	0.8	0.9	0.8	1.7	-0.3	-1.8	-0.2	-0.1
7	1	0.1	-0.5	0.6	0.3	-1.5	-0.5	-2.5	-2.9
8	1	8.7	7.7	3.6	1.7	-2.1	-1.8	-4.3	-2.8
1	2.5	5.9	9.2	5.8	4.8	-4.3	-9.7	-9.3	-8.1
2	2.5	4.7	1.2	2.5	3.4	-10.7	2	-6	-2.9
3	2.5	5.4	3.7	3.4	1.6	-7.9	-7.1	-6.5	-2.5
4	2.5	10.9	9.7	10.7	10.8	-10.4	-8.8	-10.1	-9.8
5	2.5	1.5	3.3	3.8	0.5	-2.3	-2.3	-3.3	-1.3
6	2.5	6.2	6.6	4.5	3.1	-5.5	-3.3	-3.8	-0.8
7	2.5	1.9	4.6	2	4.5	-3.1	-9.9	-5	-3.6
8	2.5	23.1	16.7	18	3.5	-12	-10.5	-18.2	-16.1

Key to Table A15 Data

- Participant – Participant ID
- GVS Amp – GVS Amplitude
  - 0 = Sham stimulation
  - 1 = 1mA GVS
  - 2.5 = 2.5mA GVS
- Slope – Slope of psychometric function for each GVS polarity and session
  - RGVS = Right-Anodal/Left-Cathodal GVS
  - LGVS = Left-Anodal/Right-Cathodal GVS
  - 1-4 = Session number

Table A15: Chapter 8, Slope Data

Participant	GVS Amp	Slope LGVS 1	Slope LGVS 2	Slope LGVS 3	Slope LGVS 4	Slope RGVS 1	Slope RGVS 2	Slope RGVS 3	Slope RGVS 4
1	0	0.6	1	0.9	1.1	0.3	1.2	1.2	0.6
2	0	1.1	1.3	1.1	1.1	0.6	3	0.7	0.4
3	0	1	0.8	0.5	0.7	0.6	0.6	0.7	1.1
4	0	4.2	2.1	1	0.7	3.3	3.3	0.6	2.8
5	0	0.1	0.5	2.2	0.5	0.7	0.4	1	0.3
6	0	1.3	1.1	0.3	0.6	0.8	0.4	0.3	0.3
7	0	33.2	1.8	0.4	0.5	1.1	3.7	1	0.6
8	0	1.4	4.2	0.9	0.7	4.5	3.3	0.6	1
1	1	3	3.4	12.1	1.4	2.5	0.5	19.6	3.5
2	1	4.1	1.3	1.2	1.1	3.7	3	1.9	1.8
3	1	1	1.9	1.3	1.7	0.5	2.1	1.4	1.5
4	1	2.1	1.5	1.5	0.8	4.2	2.4	1.8	0.3
5	1	1.6	6.9	3.8	1.7	0.5	9.4	0.8	3.8
6	1	1.4	0.7	0.6	0.8	0.5	1.7	1.4	0.6
7	1	0.9	2.4	1	2	2.3	10.8	2.6	3
8	1	7.3	5.1	3	1.1	5.5	2.2	7.2	2.1
1	2.5	8.2	7.1	7.4	16.4	3.8	4.3	9.9	5.5
2	2.5	3.9	2.7	1.7	5	13.1	16.2	15.9	6.2
3	2.5	2.1	1	1.1	2.3	2.4	2.1	4.3	0.9
4	2.5	1.9	2.5	1.7	2.1	2.3	2.2	2.4	1.4
5	2.5	9.7	5.4	9.1	1	2.6	5	4.3	2.4
6	2.5	3.1	1.4	2.2	2.1	2.9	1.9	2.4	0.3
7	2.5	2.8	4.1	1.8	6.9	1.1	8.8	5.6	2
8	2.5	8	6.8	50	4.6	7.1	9.5	8.6	14.5